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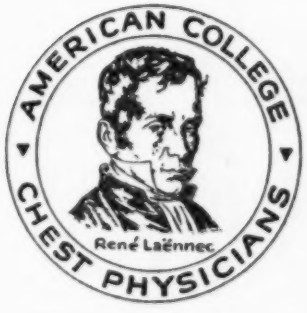

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DISEASES *of the* CHEST

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Physiologic and Antibiotic (Penicillin) Therapy in Chronic Hypertrophic Pulmonary Emphysema*

ALVAN L. BARACH, M.D., F.C.C.P., and BETTINA GARTHWAITE, M.D.**

With the technical assistance of

EVA LEVENSON, B.A., and DORA RADER, Ph.G., B.A., M.S.

INTRODUCTION

Although chronic hypertrophic emphysema of the lungs is frequently a progressive crippling disease with irreparable lesions, the abundant pessimism concerning treatment is unjustified in a considerable number of cases. The use of physiologically directed procedures, based on investigations of the pathological physiology of this condition in the past two decades, at times transforms a life of semi-invalidism to one capable of considerable activity, with comparative freedom from many of the distressing symptoms of the disease. Recent developments in penicillin therapy have provided additional techniques in the preventive and active treatment of the frequently associated chronic bronchitis.

Our purpose will be to consider pulmonary emphysema, or inflation of the lungs, as the entity characterized by a barrel-shaped chest. Senile or postural emphysema, acute emphysema and localized emphysema constitute different clinical entities.

PATHOLOGICAL PHYSIOLOGY

In presenting the pathological physiology of pulmonary emphysema, a selection of those factors that appear to be most causally

*From the Department of Medicine, College of Physicians and Surgeons, Columbia University and the Presbyterian Hospital, New York City.

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related to the production and the relief of symptoms will be made. The disease may begin after a long period of chronic bronchitis or bronchial asthma, or may quickly develop following an attack of bronchopneumonia. In either event loss of elasticity of the lungs is primarily responsible for a characteristic disturbance in the mechanics of breathing. The decrease in pulmonary elasticity, determined by measurement of the intrapleural pressure, directly portrays the tension in the lung. In pulmonary emphysema the intrapleural pressure may be the same as or near to that of the atmosphere even at the end of inspiration. Loss of lung elasticity explains over-distention of the air sacs with bullae formation which occurs on the surface of the lung. With each inspiration the superficial air cells are stretched to a larger extent than those near the hilum; repeated strain results in dilatation of the alveolar ducts and alveoli so that air no longer enters the atrium as a jet; the septa between the alveoli disappear; the supporting framework of the alveoli is lost and the amount of air in the lungs is ultimately considerably increased.[1] The pulmonary capillaries in places are ruptured and the vascular bed is diminished, with increased work on the part of the right ventricle. The increased respiratory deadspace, consisting of dilated alveoli and bullae, is exposed to irregular ventilation, and at times unrespired blood is contributed to the aortic stream from pulmonary capillaries passing through poorly ventilated enlarged air sacs.[2,3,4,5,6,7,8] The ultimate disturbance in respiratory function is the provision of a normal tension of oxygen and carbon dioxide in the arterial blood in the presence of serious impairment in the mechanism of breathing.

The arterial oxygen saturation in cases with pronounced impairment of alveolar diffusion of gases may lie in a 60 to 85 per cent range; in other patients the oxygen saturation of arterial blood may be substantially higher, from 85 to 95 per cent. The CO_2 content may be elevated 5 to 10 volumes per cent or within the normal range. Cournand and Richards[7] have emphasized the distinction between ventilatory insufficiency, or failure to provide the required pulmonary ventilation without dyspnea, and respiratory insufficiency, or failure to maintain a normal gas interchange between the alveoli and the pulmonary capillaries. A third category in their classification is combined ventilo-respiratory insufficiency; this group comprises cases that present in addition signs of cardiac failure.

Although loss of elasticity of the parenchyma of the lung may occur swiftly after certain types of pneumonic infection, the strain of chronic partial bronchial obstruction produced by bronchial spasm and bronchial infection is undoubtedly a significant contributory factor in the majority of cases. The functional pathology

which results from obstructive respiration illustrates the manner in which damage to the alveolar walls may take place.

Studies on experimental tracheal obstruction have shown that constriction of the airways during inspiration results in congestion and edema of the lungs and emphysematous changes at the periphery, whereas constriction in the airway during expiration only does not produce these pathological changes.[9,10] Similarly, inspiring against a negative pressure will produce this pulmonary pathology whereas exhaling against comparable pressures does not damage the lungs.[10] Edema in the lung may also be experimentally produced by inhaling 10 per cent oxygen against a moderate resistance which in itself does not create any pulmonary pathology, indicating the importance of anoxia in increasing the permeability of the pulmonary capillaries.[12] During inspiration through a narrowed orifice an increased negative intrapulmonary pressure takes place which is portrayed by the rise in intrapleural pressure. As obstructive dyspnea continues exudation of the serum takes place into the alveoli, with congestion and edema of the walls of the smaller bronchi. As a result of progressive increase in negative intrapulmonary pressure air is sucked into the alveoli by strong contractions of the diaphragm and chest musculature. Since the bronchi narrow during expiration the difficulty of eliminating the inhaled atmosphere is increased. The accessory muscles of the abdominal wall are then used in expiration as well as intercostals that depress the ribs.

The elastic recoil of the lungs becomes insufficient to result in efficient expulsion of the air forcibly drawn into them. Under the circumstances, therefore, of experimental tracheal obstruction the pathologically elevated negative pressure within the lungs is the cause of the patho-physiological disturbance that results. Relief of dyspnea under these circumstances has been demonstrated by inhalation of air under positive pressure, which automatically lowers the pathologically elevated negative intrapleural pressure, inhalation of helium with oxygen, which is transported at a significantly lower pressure because of the decreased molecular weight of the mixture, and by inhalation of 100 per cent oxygen, because of the lowering in pulmonary ventilation which takes place when anoxia has supervened.[8,10,11,12,13,14,15]

In patients with bronchial asthma an elevated intrapleural negative pressure in inspiration has been found, with decrease following the inhalation of helium with oxygen and atmospheres under positive pressure.[14] In this disease, before the development of pulmonary emphysema, elastic recoil of the lungs may be little or not at all impaired.

As a result of either long continued partial bronchial obstruction,

due either to infection and consequent inflammatory swelling of the smaller bronchi, or to bronchial spasm, stress on the bronchi and alveoli (the effect of repeated negative pressure) results in a loss of the peristaltic activity of the bronchial musculature and elasticity of the lung parenchyma.

When the elasticity of the lungs is finally lost, the intrapleural pressure at the height of the inspiration becomes less and less negative, and may even become positive. The elastic recoil of the lungs is then so much diminished that the accessory muscles of the abdominal wall and the intercostals are employed to squeeze air out of the lungs, which become more and more inflated with this inefficient method of emptying and ultimately distend the thorax. The diaphragm is depressed and its excursion much diminished. Contraction of the diaphragm frequently then results in an inward movement of the ribs. In addition to the effect of long continued partial bronchial obstruction, coughing, which takes place in association with bronchial infection and bronchial spasm, may exert an added strain on the pulmonary alveoli as well as the smaller bronchi and alveolar ducts.

As the diaphragm becomes a progressively impaired mechanism for ventilating the lungs inspiration is accomplished by the accessory intercostal and neck musculature. The anterior chest wall is pulled upward and forward, and the lungs are expanded to fill the space in the pleural sinuses and between the chest wall and heart. The dorsal kyphosis and increase in the anterior-posterior diameter of the chest are the result of this progressive expansion of the lungs.

The residual air is increased in most cases, with irregular ventilation of the alveoli, a decreased vital capacity and inefficient distribution of tidal air, with hypoventilation throughout a large part of the pulmonary air spaces.[7] The pulmonary ventilation, which is increased in the vast majority of cases, frequently shows an immediate reduction with inhalation of 100 per cent oxygen; in those cases that do not reveal an instantaneous lowering of the volume of breathing when pure oxygen is inhaled, residence in an atmosphere of 50 per cent oxygen for a period of one to five days generally results in a substantial decrease in ventilation.[8,16,17,18,19]

Continuous inhalation of an atmosphere of 50 per cent oxygen is followed by an increase in arterial oxygen saturation above 95 per cent and a progressive increase in arterial CO_2 content, the latter paralleling the steady fall in pulmonary ventilation. The initial response to lowering of the pulmonary ventilation by inhalation of oxygen is a rise in arterial oxygen saturation, an increase in CO_2 tension and a temporary decrease in pH. The

elevation in CO_2 tension permits an elimination of carbon dioxide with a smaller volume of ventilation, more CO_2 diffusing from the alveoli in a given volume of air because of the higher tension. The pH of the blood after a transient period of slight acidity becomes normal as a result of compensatory elimination of acid ions,[19,20] such as chlorides and others. In congestive heart failure the diuresis that takes place is accompanied by a large output of chloride ions, but in pulmonary emphysema without cardiac insufficiency the excretion of chlorides is less prominent.

In patients who manifest marked improvement with continuous oxygen therapy, the CO_2 tension in the blood tends to return toward normal even during the inhalation of 50 per cent oxygen. This improvement in respiratory function is the result of diminution in the over-distention of the lungs, decrease in bronchial inflammatory swelling, diminution in the residual air and increase in vital capacity. In those cases in which relatively slight improvement takes place in the functional pathology of the lungs the CO_2 tension remains high. That elimination of CO_2 is not the primary factor in the dyspnea of emphysema may be seen by the response to continuous inhalation of oxygen, namely relief of dyspnea, even in the presence of an *increased* arterial CO_2 content and CO_2 tension; the significant disturbance in respiratory function is the inability to diffuse an adequate supply of oxygen from the alveoli into the lungs. This opinion is not invalidated by the fact that some patients with pulmonary emphysema show a normal arterial oxygen saturation, since inhalation of oxygen in these same patients generally results in relief of dyspnea. Patients with pulmonary emphysema may maintain a burdensome increase in pulmonary ventilation which itself produces a normal or nearly normal arterial oxygen tension. Proprioceptive reflexes from the lungs and chest wall are responsible for the transmission intracerebrally of the sensation of dyspnea and the degree of dyspnea is apt to vary with the extent to which the pulmonary ventilation approaches the maximal ventilation or the vital capacity[6,7] but the increased respiratory effort which is perceived as labored breathing does actually maintain a constancy of the internal environment in respect to tissue oxygen supply, and a restoration of a plentiful oxygen supply is accompanied in the large majority of these cases with a sufficiently decreased pulmonary ventilation as to result in relief of dyspnea.[8,15]

In 7 of 10 patients with chronic pulmonary disease previously studied, the CO_2 content of the arterial blood rose 20 vol. per cent during the inhalation of 50 per cent oxygen.[8b] This was accompanied by relief of dyspnea and decrease in pulmonary ventilation. In some patients in whom 100 per cent oxygen did not result in

an immediate fall in pulmonary ventilation the relief of bronchial spasm by inhalation of 1:100 epinephrine and neosynephrine was subsequently followed by lowering of the pulmonary ventilation when 100 per cent oxygen was inhaled.[8,15]

Since the mechanical difficulties of breathing in patients with pulmonary emphysema have been traced to the loss of pulmonary elasticity, the over-inflated lung and the flattened position of the diaphragm, attempts were made to direct pressure on the abdomen in order to press the leaves of the diaphragm upward and, thus having forced the diaphragm to its normal position of relaxation, it was capable of moving downward again and diaphragmatic breathing was thereby restored in a limited way. This observation led to the designing of the abdominal belt.[4] In 12 patients who have been recently studied repeated compression of the upper abdomen inward and upward during the latter third of expiration resulted in an increase in vital capacity of 200 to 1000 cc. In those patients who do not rapidly trap air again this is accompanied by a decrease in pulmonary ventilation and relief of dyspnea.

PHYSIOLOGIC TREATMENT OF PULMONARY EMPHYSEMA

The treatment of bronchial obstruction in pulmonary emphysema is frequently overlooked as a method of profound importance in diminishing the patho-physiological disturbance in this condition. The program of bronchial relaxation used in asthma may be applied with certain modifications.[21] Partial bronchial obstruction is produced not only by bronchial spasm but also by an excessive production of mucus, which may be formed partly as the result of an allergic reaction to dust and other allergens, but for the most part as a response to infection. In a consideration of the treatment of pulmonary emphysema, measures which diminish bronchial infection and bronchial spasm are now of special value. However, even when the condition has become well established it is still pertinent to inquire whether allergic factors, such as dust, may be involved in maintaining bronchial spasm. The search for an adequate control of infection was instituted shortly after studies of inhalation of penicillin aerosol began.[22]

Although cases of chronic bronchitis have been treated with penicillin with favorable results, especially those in which hemolytic streptococcus or pneumococcus organisms were found on culture, many patients with pulmonary emphysema and chronic bronchitis have been markedly benefited by physiologically directed treatment.[8,15,18,19,20,23] In some of the cases who were referred for penicillin therapy the employment of measures to promote bronchial relaxation, oxygen therapy, as well as other forms of physiologic therapy, produced sufficiently good clinical results as to

render unnecessary the trial of antibiotic measures. Whether or not patients with pulmonary emphysema show signs of bronchial spasm by examination of the lungs, such as sibilant rales, the presence of prolonged expiration is an indication for the use of measures which produce bronchial relaxation.[21]

Although epinephrine and ephedrine have been recognized as valuable in the treatment of bronchial spasm in patients with this disease the effective employment of aminophyllin has received little attention. The administration of aminophyllin by mouth on an empty stomach is a valuable method of producing relaxation of the bronchi in a considerable proportion of patients with pulmonary emphysema, frequently resulting in marked decrease in cough, expectoration and exertional dyspnea. The dosage generally employed is 0.2 gram immediately on arising and again at 3 to 4 in the afternoon. In some cases the morning dose is 0.3 gram if the patient can tolerate this amount without nausea. The administration of aminophyllin in larger dosages may be given rectally by dissolving 0.6 gram in 25 cc. of water and instilling it with a glass syringe and catheter,[24] or 0.3 to 0.5 gm. may be given as a suppository. When the symptoms due to bronchospasm are relieved, it is desirable to stop aminophyllin for variable periods, five days to a week or more, to prevent the patient from becoming refractory to its bronchodilator effect.

Ephedrine may be found preferable in some patients to aminophyllin and may be given in a dosage of 25 mgm. on arising, after breakfast and at 3 p. m. It is generally not prescribed at night because it may maintain a wakeful state, although this effect may be counteracted when administered with 75 mgm. of amytal or 0.1 gm. phenobarbital. In patients who have become refractory to aminophyllin, ephedrine is substituted, and in those patients who respond less and less well to ephedrine, aminophyllin is employed. In many cases refractoriness to ephedrine and aminophyllin does not develop and under these circumstances almost complete disappearance of cough and marked improvement in dyspnea take place when a capsule containing aminophyllin 0.2 or 0.3 gm. combined with ephedrine, 25 mgm., is administered on arising and at 3 p. m.

In a small proportion of cases oral administration of aminophyllin and ephedrine does not yield as much benefit as the inhalation of a mixture of 0.3 cc. of 1:100 epinephrine with 0.5 cc. of 1 per cent neosynephrine, nebulized by a flow of 5 liters per minute of oxygen[25] and continuously inhaled in a nebulizer of fine particle size* or in a penicillin rebreathing nebulizer apparatus.[22,27]

*The nebulizer and solution made by the Vaponefrin Co., Upper Darby, Pa., is suitable for this purpose.

The use of 1:75 or 1:100 epinephrine by the hand bulb is also of considerable value in these patients, especially when two or three inhalations are taken in the morning and at 3 to 4 in the afternoon and during the night if coughing takes place.

The inhalation of nebulized epinephrine should not generally be relied upon exclusively since patients are apt to use it more and more constantly with the prospect of refractoriness to the drug developing more rapidly. A combination, therefore, of periodic administration of aminophyllin by mouth and inhalation of epinephrine, or ephedrine by mouth and inhalation of epinephrine, is generally recommended.

The development of progressive lack of response to bronchodilator drugs in pulmonary emphysema presents a difficult problem but one that can usually be handled more easily than in patients with intractable bronchial asthma. The simplest method of restoring sensitiveness to bronchodilator drugs in patients with pulmonary emphysema is bed rest and the administration of demerol, which usually produces bronchial relaxation. If the patient is treated at home, 50 mgm. by mouth may be given after each meal (or two meals) and before going to bed. Although a few patients may become unpleasantly groggy or nauseated by it, the majority do not show sufficient disturbance to lower this dosage. Loss of appetite may be encountered but it quickly returns when the drug is stopped. If the patient is in the hospital or has a nurse, hypodermic administration of 50 mgm. of demerol after breakfast, at 3 p. m. or after dinner and on retiring may be employed. During this period of relaxation with demerol, ephedrine and aminophyllin should be stopped or the dosage reduced to once a day on arising; less use is made of the epinephrine nebulizer.

After a period of 4 to 6 days it is generally found that restoration of sensitiveness to both aminophyllin and ephedrine has taken place, except in more severe cases. It is often of value to decrease the dosage of demerol during the last three days of the week to 50 mgm. after dinner and 50 mgm. on retiring. Special emphasis should be given to the prescription of rest at this time. If the patient is not at complete bed rest instructions to lie down for an hour and one-half after each dose of demerol should be emphasized. The possibility of demerol addiction should be kept in mind and the length of its use restricted for the most part to four to six days. After a week of this type of therapy aminophyllin and/or ephedrine may then be continued and the patient may become ambulatory. Oxygen therapy, which may be profitably used at the same time, will be discussed in detail below.

In addition to bronchial relaxation therapy, physiological procedures that aim to produce relief of the mechanical insufficiency

of the diaphragm should be carried out. In patients who have a moderately full abdomen the elastic belt should be tried and worn during the day. Of greater value in most instances is the procedure which aims to employ the overdistended lungs by mechanical elevation of both leaves of the diaphragm through manual compression.[28] This maneuver is at first done by the doctor and then explained to the patient so that he may perform it on himself. The palms of both hands are placed *underneath* the ribs, not over the lower ribs, and during the latter third of expiration the hands are pushed inward and upward. The trapped air within the lung may be heard as it is expelled through the narrowed bronchi, frequently as a wheeze. When this procedure has been performed 10 or 15 times the patient is frequently in a much improved state in respect to dyspnea. The vital capacity may be substantially elevated and fluoroscopic observation reveals a diaphragm that now moves much more readily and over a wider excursion than previously. This procedure of manual compression of the upper abdomen underneath the ribs during the latter part of expiration is recommended three times daily, generally only for one or two minutes and, in addition, at any time when the patient may develop an acute functional over-inflation of his lungs as the result of exertion or cough. Better results are often produced when inhalation of 1:100 epinephrine is carried out with a hand nebulizer immediately preceding.

Inhalation of 70 to 100 per cent oxygen in a mask for 20 to 30 minutes two or three times a day is an additional help by lowering the volume of ventilation and by decreasing the degree of functional over-inflation of the lungs. The physiological basis for this procedure is the longer time available for emptying the alveoli when breathing is slower. In a considerable number of patients the temptation to use 100 per cent oxygen more frequently is a real one, especially in those who have a mechanism that traps air very quickly. It is better to limit inhalation of 100 per cent oxygen for 20 to 30 minutes to 2 or 4 times daily than to use this concentration continuously.

In a considerable proportion of patients continuous oxygen therapy may be employed with expectation of marked improvement in dyspnea and a restoration of a more normal functioning of the bronchi and lungs after treatment has been discontinued. Since there are other patients with pulmonary emphysema in whom inhalation of oxygen can be abandoned only with recurrence of severe dyspnea, the selection of cases as well as the technique is of considerable importance. Senile or postural emphysema, in which the chest is not enlarged in its anterior-posterior diameter, is the form of this disease in which little or no improvement may

persist, after oxygen treatment has been discontinued. Patients with intense over-distention of the alveoli may also manifest improvement only during the course of continuous oxygen therapy and not afterward. However, patients with pulmonary fibrosis, pulmonary emphysema and congestive heart failure may respond remarkably to continuous inhalation of oxygen, and with considerable maintained improvement, if a gradual lowering of the oxygen concentration is carefully carried out. In the presence of congestive heart failure, in association with pulmonary emphysema, rigid restriction of sodium in all forms (sodium chloride and bicarbonate, etc.) is required although no restriction of fluids is necessary, during and after the program of oxygen treatment. The patient with pulmonary emphysema who is in severe respiratory difficulty at rest poses a problem in respect to deciding whether to use continuous oxygen treatment. Since the relief of dyspnea that is obtained as the result of continuous inhalation of 50 per cent oxygen may be partially or at times wholly lost if oxygen therapy is entirely discontinued, it should generally not be administered to patients with senile or postural emphysema or to patients with intense over-distention unless skilled medical management is available as well as intermittent oxygen therapy feasible at home following its use continuously. The technique of oxygen treatment in cases of pulmonary emphysema depends on the form of the impairment in respiratory function.

In patients who have chronic arterial anoxia, manifested by cyanosis, the administration of oxygen should be begun with relatively low concentrations. When these patients are abruptly exposed to 50 per cent oxygen irrationality and delirium may take place in three to twelve hours and may last from two to seven days if oxygen treatment is continued, and then spontaneously disappear.[8,15,19,20,29] When the percentage of oxygen in the atmosphere is gradually increased from 21 to 50 per cent, no such impairment in mental functioning is produced. The patient may be started on nasal catheter oxygen, 1 liter per minute for two days, increasing 1 liter per minute each day, until 5 or 6 liters per minute are inhaled. The rubber catheter may be placed in the nasal or oral pharynx, or double bent soft rubber tubes[30] may be used with greater comfort. The ends of the curved nasal tubes enter the nostril for a distance of one-half inch, similar to that of the metal forked nasal tube. When an oxygen flow of 5 to 6 liters per minute is reached, this oxygen-enriched atmosphere, approximately 35 to 37 per cent oxygen if the catheter is in the nasopharynx and 40 to 45 per cent oxygen if it is placed in the oropharynx, may be inhaled for a period of one to four weeks, or longer, if desired. If the condition of the patient indicates adequate restoration of

respiratory function and relief of dyspnea, the oxygen concentration may be gradually lowered by reducing the flow 1 liter a minute every one or two days.

If opportunity is available for measuring the arterial CO_2 content (or the plasma bicarbonate of venous blood), the indications for lowering the oxygen concentration may be more clearly determined. After an initial rise in arterial CO_2 content from 50 to approximately 75 to 80 vol. per cent, in some cases 90 to 100 vol. per cent, the CO_2 content in arterial blood may gradually fall 10 or 15 vol. per cent. Since this is an indication of improvement in physiological functioning of the lungs and bronchi, the oxygen concentration may then be safely lowered 1 liter every two days until the patient is removed from an atmosphere of oxygen entirely. In these cases it is generally advisable to provide inhalation of 100 per cent oxygen for 20 minutes two or three times a day. In a few patients the administration of oxygen for longer periods of time may be carried out depending upon the patient's clinical indication and social and economic factors.

If the patient with pulmonary emphysema has no pre-existing chronic arterial anoxia as evidenced by cyanosis, inhalation of 50 per cent oxygen may be begun without preliminary gradual increase in oxygen concentration. An oxygen tent may be employed or a nasal catheter or a nostril inhalation apparatus. The oxygen mask is generally not suitable for continuous therapy in patients with pulmonary emphysema. Subjective discomfort and psychic elevation of the pulmonary ventilation are apt to take place and control of gradual decrease in oxygen concentration is difficult; the nasal catheter and bent nasal tubes are more practical methods for this purpose. After a course of oxygen therapy has been satisfactorily completed, the measures described above to maintain bronchial relaxation are employed. The patient is instructed to avoid physical exertion that brings about dyspnea and consequent over-inflation of the lungs. Deep breathing exercises are hazardous because trapping of air generally follows arbitrary increase in the volume of breathing, no matter how it is induced. When the lungs have been severely over-distended by undue physical effort, another course of rest, demerol and oxygen may be required to restore adequate pulmonary function.

ANTIBIOTIC (PENICILLIN) THERAPY

Antibiotic therapy with penicillin is indicated in cases in which bronchial or broncho-alveolar infection is present. The administration of penicillin by inhalation as an aerosol was especially used in this study, although in some cases penicillin was given intramuscularly and by mouth as will be seen in the accompanying

TABLE 1
PULMONARY EMPHYSEMA TREATED WITH PENICILLIN AND PHYSIOLOGICALLY DIRECTED THERAPY

Case No.	Sex and Age	Additional Diagnosis	Course	Route of Administration and Total Dosage		Duration of Therapy (days)	Improvement Due to Penicillin Therapy	Improvement Due to Physiologically Directed Therapy	Duration of Improvement Less than 2 months	Duration of Improvement More than 2 months
1	M 36	Asthma, chronic bronchiolitis	1	I.	1,060,000	9	marked	moderate	+	
2	F 61	Asthma, pulmonary fibrosis	1	I.	660,000	7	marked	moderate		+
			2	I.	1,300,000	7	none	slight		
				I.M.	470,000	14				
			3	I.	3,400,000	16	none	none		
				I.M.	6,426,000	11				
3	F 40	Asthma, chronic bronchiolitis, pulmonary fibrosis	1	I.	1,400,000	7	moderate	moderate	+	
4	M 52	Pulmonary fibrosis, cardiac insufficiency	1	I.	840,000	7	slight	slight		
5	F 60	Asthma, chronic bronchiolitis	1	I.	2,500,000	8	marked	moderate	+	
			2	I.	2,000,000	10	marked	slight		+
6	F 60	Asthma, pulmonary fibrosis	1	I.	2,020,000	12	marked	slight	+	
			2	I.	1,300,000	7	moderate	slight	+	
			3	I.M.	1,050,000	11	moderate	moderate	+	
			4	S.C.	1,500,000	5	none	moderate	+	
7	F 63	Asthma, chronic bronchitis, chronic sinusitis	1	I.	1,770,000	10	moderate	moderate	+	
				I.M.	840,000	7				
			2	I.M.	1,230,000	11	slight	moderate	+	
				I.M.&S.C.	2,250,000	10				
			3	O.	3,800,000	9	slight	marked	+	
			4	I. (NS)	300,000	3	slight	moderate	+	
				I.M.	500,000	3				
8	F 42	Asthma, chronic bronchitis, chronic sinusitis	1	I.	1,500,000	10	moderate	marked		+
			2	I.	300,000	3	slight	moderate	+	
				I.M.	5,392,500	25				
			3	I.	3,000,000	21	moderate	slight	+	
			4	I. (NS)	1,000,000	10	marked	slight		+
			5	I.	2,125,000	17	marked	slight		+
				I.M.	3,400,000					
			6	I.	3,000,000	21	moderate	slight	+	
9	F	Bronchiectasis,	1	I.	950,000	5	marked	moderate	+	

9	F 56	Bronchiectasis, pulmonary fibrosis	1 2 3 4 5	I. I. I.M. I. S.C. O. I.	950,000 2,300,000 600,000 2,440,000 1,180,000 60,000,000 500,000	5 13 8 12 4 90 3	marked marked slight none none	moderate moderate marked moderate moderate	+ + + + +	
10	F 71	Asthma, chronic bronchitis, chronic sinusitis	1 2	I. I. (NS)	2,050,000 6,000,000	8 70	marked moderate	moderate moderate		+ +
11	M 21	Bronchiectasis, asthma	1 2 3 4 5	I. I.M. I. I. I.M. O. I.	600,000 700,000 1,800,000 1,000,000 1,080,000 8,000,000 50,000 U 4x daily	3 8 12 8 14 1 yr.	moderate none slight none marked	slight none moderate none none	+ + 	 +
12	M 49	Asthma	1 2	I. O.	2,200,000 3,000,000	12 4	moderate none	moderate none	+	
13	M 60	Asthma, chronic sinusitis	1	I. I.M.	2,440,000 370,000	14 5	none	moderate		+
14	M 33	Asthma, chronic sinusitis	1 2	I. (NS) I. (NS)	4,000,000 2,100,000	20 14	marked marked	slight none	+ 	 +
15	M 47	Asthma, bronchiectasis, pulmonary fibrosis, infectious arthritis	1 2 	I. I.M. I. O. I.M.	540,000 1,155,000 3,250,000 3,300,000 7,650,000	5 10 60 	none none 	slight slight 		
16	M 69	Bronchiectasis, chronic bronchitis, chronic sinusitis	1	I.	1,300,000	7	moderate	none		+
17	M 62	Bronchiectasis, chronic sinusitis, pulmonary fibrosis	1	S.C. I.M. I.	1,260,000 2,230,000 2,550,000	8 11 12	marked	slight		+
18	M 33	Pulmonary fibrosis, chronic pneumonitis	1	I. I.	650,000 2,625,000	5 17	slight	moderate		+

I. — Oral inhalation.
I.M. — Intramuscular injection.
S.C. — Subcutaneous injection.
O. — Oral ingestion.
I. (NS) — Nasal inhalation with intermittent negative pressure.

TABLE 1 (Continued)

PULMONARY EMPHYSEMA TREATED WITH PENICILLIN AND PHYSIOLOGICALLY DIRECTED THERAPY

Case No.	Sex and Age	Additional Diagnosis	Course	Route of Administration and Total Dosage	Duration of Therapy (days)	Improvement Due to Penicillin Therapy	Improvement Due to Physiologically Directed Therapy	Duration of Improvement: Less than 2 months	Duration of Improvement: More than 2 months
19	M 46	Asthma, chronic sinusitis, chronic bronchitis	1	I. 6,766,700 S.C.&I.M. 6,760,000	33 21	slight	moderate	+	
			2	O. 11,200,000	14	none	moderate		+
20	M 67	Asthma, bronchiectasis	1	I. 3,000,000 I.M.&S.C. 2,800,000	15 12	slight	moderate		+
21	M 60	Asthma, bronchiectasis	1	I. 1,300,000 I.M. 1,000,000	7	moderate	moderate	+	
			2	I. 2,000,000	14	slight	moderate	+	
			3	I. 1,000,000 (Crystalline)	10	slight	slight		
22	M 53	Asthma, chronic sinusitis	1	I. 5,560,000 I.M. 750,000	14 3	slight	moderate		+
23	F 60	Pulmonary fibrosis, bronchopneumonia	1	I. 1,300,000 O. 5,700,000	7 10	marked	none		+
24	F 67	Pulmonary fibrosis, chronic bronchitis, bronchopneumonia	1	I. 1,750,000 O. 1,700,000	10	marked	none		+
25	F 44	Asthma, allergic rhinitis, acute sinusitis	1	I. (NS) 950,000 I.M. 1,900,000	13 14	slight	moderate	+	
26	F 46	Asthma	1	I. 2,000,000 O. 3,550,000 I.M. 1,800,000	10 5 6	none slight			
27	M 54	Pulmonary fibrosis, chronic bronchitis	1	I. 1,500,000 O. 4,000,000	9	none	moderate		+
28	F 53	Asthma, acute bronchitis	1	O. 3,300,000 I.M. 1,300,000	4 10	moderate	moderate		+

29	F 63	Asthma, chronic bronchitis, pulmonary fibrosis, chronic sinusitis	1	I. O.	3,600,000 1,800,000	9	slight	moderate		+
30	F 55	Chronic bronchitis, bronchiectasis, chronic sinusitis	1 2 3 4	O. O. I. (NS) O.	6,000,000 7,000,000 500,000 5,000,000	11 11 21 11	marked moderate moderate moderate	none none none none	+ + +	 +
31	M 41	Asthma, chronic sinusitis	1	I. (NS)	1,550,000	10	none	none		
32	M 68	Asthma, chronic sinusitis	1	I. (NS) O.	2,500,000 2,000,000	10	none	none		
33	M 66	Pulmonary fibrosis, chronic sinusitis, chronic bronchitis	1 2	I. I.M. I. (NS)	1,000,000 2,075,000 1,400,000	10 15 7	moderate moderate	marked marked		+ +
34	F 68	Asthma, chronic sinusitis	1	I. (NS) O.	1,000,000 15,150,000	12 19	none	moderate		+
35	F 54	Asthma, acute pharyngitis	1	O.	5,900,000	9	slight	marked		+
36	M 70	Chronic bronchitis, chronic sinusitis	1	I. O.	2,650,000 4,900,000	6 9	slight	slight		
37	F 57	Asthma	1	I.M.	650,000	8	none	none		
38	F 36	Asthma, chronic bronchitis, chronic sinusitis, periarteritis nodosa	1 2	I. (NS) O. I.M. I. I.M.	4,000,000 3,000,000 1,500,000 1,140,000 680,000	23 10 12 10 5	slight none	slight none		
39	F 54	Pulmonary fibrosis, bronchiectasis	1	I.	2,650,000	12	none	slight		
40	M 54	Asthma, chronic sinusitis	1	I.	650,000	4	none	slight		

I. — Oral inhalation.
I.M. — Intramuscular injection.
S.C. — Subcutaneous injection.
O. — Oral ingestion.
I. (NS) — Nasal inhalation with intermittent negative pressure.

TABLE 1 (Continued)

PULMONARY EMPHYSEMA TREATED WITH PENICILLIN AND PHYSIOLOGICALLY DIRECTED THERAPY

Case No.	Sex and Age	Additional Diagnosis	Course	Route of Administration and Total Dosage		Duration of Therapy (days)	Improvement Due to Penicillin Therapy	Improvement Due to Physiologically Directed Therapy	Duration of Improvement	
									Less than 2 months	More than 2 months
41	F 5	Asthma, chronic pneumonitis	1	I.	2,800,000	20	moderate	moderate	+	
			2	I.M.	1,600,000	13	slight	moderate		+
				I.	4,000,000	26				
42	F 31	Asthma, chronic sinusitis	1	I. (NS)	750,000	8	slight	moderate	+	
			2	I. (NS)	3,000,000	21	slight	slight		
				I.	1,000,000	10				
43	F 63	Pulmonary fibrosis	1	I.	1,920,000	32	slight	marked		+
44	M 50	Pulmonary fibrosis, chronic sinusitis	1	I. (NS)	1,600,000	13	moderate	marked		+
				I.	400,000	9				
45	M 57	Asthma, bronchiectasis	1	I.	1,600,000	8	slight	moderate		+
				I.M.	675,000	3				
46	M 71	Bronchiectasis	1	I.	1,250,000	5	marked	none		+
47	M 50	Asthma, chronic bronchitis, chronic sinusitis	1	I. (NS)	3,000,000	19	none	slight		
48	M 70	Chronic bronchitis	1	I.	300,000	3	slight	moderate		+
				O.	3,500,000	7				
49	F 55	Asthma	1	I.	1,000,000	8	slight	moderate		+
50	M 33	Asthma, chronic sinusitis, chronic bronchitis	1	I. (NS)	5,000,000	15	marked	moderate	+	
51	M 64	Asthma, chronic bronchitis, chronic sinusitis	1	I.	6,000,000	30	marked	marked		+

I. — Oral inhalation.
I.M. — Intramuscular injection.
S.C. — Subcutaneous injection.
O. — Oral ingestion.
I. (NS) — Nasal inhalation with intermittent negative pressure.

tables (Tables 1 and 2).* The 51 patients on whom results are reported suffered from pulmonary emphysema as a predominating condition, but had associated with it in the majority of cases bronchial asthma and in a small number bronchiectasis, pulmonary fibrosis, sinusitis, and/or bronchitis. The principles and techniques of penicillin administration by inhalation in this study have been recently presented in papers dealing with its effect in patients with asthma, sinusitis, chronic bronchitis, bronchiectasis and lung abscess and, therefore, will be briefly referred to in this paper.[31,32,33,34,27,22] The dosage, duration of penicillin treatment and the method of administration for each patient in this study is presented in Table 1.

Nebulization of penicillin is generally accomplished by employing a stream of oxygen from a high pressure cylinder through a nebulizer of the Vaponefrin type that produces particles of relatively small size. Air from a suitable pump or compressor may also be used. Recently, we suggested the use of an automobile foot pump which can be operated by the patient at a very considerable decrease in cost.** With the oxygen method of operation, a Y tube is attached to the rubber connection between the regulator and the nebulizer; the open end is closed by a finger during the inspiratory cycle, allowing oxygen to escape during expiration and thus manufacturing penicillin only during the inspiratory cycle. If the patient were able to hold his breath for several seconds after the inhalation better deposition of penicillin on the bronchoalveolar surface would take place. Since approximately 50 per cent of the aerosol passes out into the atmosphere during expiration, a rebreathing bag was attached to the mouthpiece connected with the nebulizer to permit additional opportunity for reinhalation of penicillin. A flow of 6 to 10 liters per minute of oxygen may be employed. The development of crystalline penicillin made possible the use of a clear, odorless and non-irritating preparation which also results in higher blood levels than the conventional sodium or calcium salt. The drug is generally dissolved in normal saline, a concentration of 50,000 units per cc. being used; in some cases it is better to employ 20,000 units per cc.

When patients are too ill to cooperate with the Y tube arrangement described above, the nebulizer may be attached directly to the oxygen orifice of an oral-nasal Meter mask. In this instance

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*Intramuscular injection of two daily doses of *Crystalline* penicillin, 300,000 to 400,000 units in 4 to 5 cc. normal saline, is now being used in some of these cases.

**Barach, A. L., Rumsey, C. C., and Rader, D.: "A Simplified Technique of Treating Sinusitis with Penicillin Aerosol with a Description of a Foot Pump for Economical Nebulization of Penicillin and Other Therapeutic Aerosols," *New York State J. Med.* (To be published).

TABLE 2

PULMONARY EMPHYSEMA TREATED WITH PENICILLIN AND PHYSIOLOGICALLY DIRECTED THERAPY

Case No.	Course	SPUTUM CULTURES *Before Treatment*	SPUTUM CULTURES *After Treatment*	REACTIONS	REMARKS
1	1	Strep. viridans	0	Few urticarial spots, not persisting.	Marked improvement previously obtained by physiologic therapy.
2	1	Pneumococcus, type 31	No pneumococcus	0	Improvement lasted 2½ months after 1st course. Later, benefited from sinus treatment with aerosol and negative pressure.
	2	Strep. viridans	Strep. viridans	0	
	3	Strep. viridans	B. pyocyaneus	Increased cough at start of inhalations.	
3	1	Pneumococcus, type 3	No pneumococcus	0	Recurrence in 10 days, but good improvement on continued therapy at home.
4	1	Hemolytic Strep. Strep. viridans	Strep. viridans	0	Marked improvement previously obtained by physiologic therapy.
5	1	Strep. viridans	0	0	Dramatic improvement after 2nd course for over 1 year.
	2	Strep. viridans	B. aerogenes	0	
6	1	Gram positive diplococci	B. coli	0	Marked improvement for 1 year previously obtained by physiologic therapy. Later, frequent relapses.
	2	0	0	0	
	3	0	0	0	
	4	0	0	Local at sites of injections (5% glucose with adrenalin used as diluent).	
7	1	Strep. viridans	B. coli Strep. viridans	Increased cough on sodium salt.	Chronic bronchial obstruction, bronchospasm and infection. Respiratory invalid.
	2	Friedlander's bacillus Strep. viridans B. pyocyaneus	Hemolytic B. pyocyaneus	Local at sites of injection (5% glucose and adrenalin used as diluent). Urticaria.	
	3	B. pyocyaneus B. coli	B. pyocyaneus		
	4	Strep. viridans Staph. aureus	0	Increase in asthma.	Expired following persistent status asthmaticus less than 2 years after 1st course of therapy.

8	1	Strep. viridans	B. coli	0	Emphysema largely funtional, but severe during status asthma.
	2	Staph. aureus Yeasts	B. aerogenes	0 0	
	3	0	0	0	
	4	0	0	0	
	5	0	0	0	
	6	0	0	0	
9	1	Strep. viridans	Strep. viridans	0	Physiologic therapy, mainly oxygen, produced marked relief from cough and dyspnea, not maintained at home.
	2	Strep. viridans	Strep. viridans	Pruritis.	
	3	No growth	B. aerogenes	Local at sites of injection (5% glucose with adrenalin used as diluent).	
	4	0	0	0	
	5	Strep. viridans	B. coli	0	
10	1	Hemolytic Staph. aureus	Gram negative bacillus	0	Previous course of I.M. penicillin ineffective. Further improvement in all symptoms on aerosol treatment with negative pressure.
	2	Strep. viridans	0	0	
11	1	Strep. viridans	0	0	Remarkable benefit on continuous 1 year program. Gain of 35 lbs. in weight. Respiratory function markedly improved.
	2	Strep. viridans	B. aerogenes	Sore, reddened throat on sodium salt; pulmonary edema.	
	3	0	B. aerogenes	Urticaria and edema of the ankles.	
	4	Strep. viridans	B. aerogenes	0	
	5	Strep. viridans Staph. aureus	B. aerogenes B. coli	0	
12	1	Strep. viridans	0	0	Marked initial improvement with relapse later.
	2	0	0	Increased asthma.	
13	1	0	B. coli	Increased asthma.	Initial marked improvement with recurrence of asthma due to penicillin or sulfonamide sensitivity.
14	1	Strep. viridans	B. aerogenes	0	Emphysema largely funtional, but severe during status asthma. Marked improvement in asthma with clearing of sinusitis.
	2	0	0	0	

TABLE 2 (Continued)
PULMONARY EMPHYSEMA TREATED WITH PENICILLIN AND PHYSIOLOGICALLY DIRECTED THERAPY

Case No.	Course	SPUTUM CULTURES Before Treatment	After Treatment	REACTIONS	REMARKS
15	1	Strep. viridans	B. aerogenes B. coli	Increased cough on sodium salt.	Relieved of status asthma, but moderate asthma persisted.
	2	Strep. viridans	B. aerogenes	Soreness at injection sites (with adrenalin added to penicillin).	
16	1	Pneumococcus, type 12	B. coli	0	Decrease in cough and expectoration.
17	1	Strep. viridans	B. aerogenes	Soreness at injection sites.	Improvement maintained on 50,000 U penicillin aerosol twice daily for 1 year.
18	1	Hemolytic B. pyocyaneus	B. aerogenes	0	Marked improvement in respiratory function.
19	1	Pneumococcus, type 19 Strep. viridans	B. aerogenes	Local at injection sites (5% glucose with adrenalin used as diluent).	Relieved of status asthma, ambulatory, but asthma persists. Emphysema largely functional.
	2	Strep. viridans B. proteus	0	0	
20	1	Strep. viridans	B. aerogenes B. coli	Local at injection sites (5% glucose with adrenalin used as diluent).	Asthma persists, but improved.
21	1	Strep. viridans	B. coli	Urticaria, fever, exacerbation of asthma.	Asthma persists, but improved.
	2	Strep. viridans	B. aerogenes	Sore, reddened tongue, exacerbation of asthma.	
	3	Non-hemolytic Strep.	B. coli	Increased dyspnea and wheezing.	
22	1	Hemolytic B. aerogenes Staph. albus	B. aerogenes B. coli	Urticaria and exacerbation of asthma.	Asthma persists, but improved.
23	1	Pneumococcus, type 17	B. aerogenes	0	Strikingly improved with decrease in cough and expectoration.
24	1	0	B. coli** B. aerogenes**	0	Rapid symptomatic improvement with almost complete clearing of basal rales on penicillin therapy.

** Throat culture.

25	1	Strep. viridans	B. aerogenes	Exacerbation of asthma.	Oxygen and helium inhalations combined with usual broncho-dilator drugs afforded chief relief. Breathing exercises helped moderately.
26	1	Strep. viridans	B. aerogenes	Nausea from oral penicillin.	Slight and temporary relief from bronchial relaxation. Fever therapy with triple typhoid vaccine broke status asthma; occasional attacks controlled by broncho-dilator drugs.
27	1	Strep. viridans	B. aerogenes	0	Cough and expectoration decreased, but patient became dyspneic during penicillin inhalations.
28	1	Strep. viridans	Strep. viridans	Soreness at injection sites (adrenalin added to penicillin).	Asthma less severe following control of infection.
29	1	Strep. viridans	B. aerogenes	0	Chief benefit was from physiologic therapy.
30	1	Staph. albus Strep. viridans	0	Coated brown tongue and stained teeth.	Exacerbation of symptoms following acute bronchitis. Rales at posterior lung bases cleared after therapy. Symptoms controlled fairly well by single occasional inhalations.
	2	H. hemolyticus H. influenzae B. coli	B. coli H. influenzae B. aerogenes	" "	
	3	Hemolytic Strep.	B. coli	"	
	4	0	0	"	
31	1	Strep. viridans	B. coli	Increased wheezing following inhalations.	Symptoms controlled on penicillin but returned when therapy stopped. Previously responded well to physiologic therapy.
32	1	Strep. viridans Hemolytic Strep.	B. aerogenes	Increased wheezing following inhalations.	Some control of infection but no improvement in asthma.
33	1	0	B. aerogenes	0	Marked improvement after oxygen therapy. Penicillin aided in controlling infection.
	2			0	Marked improvement from I.M. beeswax penicillin (2 courses) at home.
34	1	B. alkaligenes	0	Sore, reddened tongue.	Intractable asthma responded to physiologic therapy despite no improvement in sinusitis on penicillin therapy.

TABLE 2 (Continued)

PULMONARY EMPHYSEMA TREATED WITH PENICILLIN AND PHYSIOLOGICALLY DIRECTED THERAPY

Case No.	Course	SPUTUM CULTURES Before Treatment	SPUTUM CULTURES After Treatment	REACTIONS	REMARKS
35	1	Strep. viridans	B. aerogenes	0	Intractable asthma for several months responded to filtered air prior to use of penicillin. No broncho-dilator drugs required after 5th day.
36	1	No growth	B. aerogenes B. coli	Sore, reddened tongue and throat, increased cough.	Rales cleared on penicillin therapy and aminophyllin.
37	1	0	0	Urticaria, soreness at injection sites (adrenalin added to penicillin).	Intractable asthma failed to respond to usual physiologic therapy, filtered air, and ether anesthesia. Finally improved.
38	1	Hemolytic B. pyocyaneus	B. aerogenes B. coli	Exacerbation of asthma.	Intractable asthma for 3 years. Temporary improvement in hospital following fever due to bronchopneumonia. Autopsy diagnosis: Periarteritis nodosa.
	2	Hemolytic B. pyocyaneus	B. aerogenes	0	
39	1	0	B. coli B. aerogenes	Urticaria.	No change in pulmonary condition. Died from abdominal complications on later admission.
40	1	Hemolytic Strep.	B. aerogenes	Urticaria, exacerbation of asthma.	Previously markedly improved for 3 months following I.M. penicillin. Helped mainly by physiologic therapy at present.
41	1	Hemolytic Staph. aureus	0	0	Clinical improvement without change in x-rays. Chief benefit from physiologic therapy although penicillin useful in controlling acute infection (organism moderately resistant to penicillin).
	2	Hemolytic Staph. aureus	H. influenzae	0	

42	1	0	B. aerogenes Staph. aureus	0	Chief benefit from physiologic therapy. Sinusitis unimproved.
	2	0	B. aerogenes	0	Only temporary improvement, chiefly from physiologic therapy.
43	1	Strep. viridans	Strep. viridans	0	Marked improvement from oxygen therapy.
44	1	Strep. viridans Hemolytic Strep. Staph. aureaus	Yeasts	Irritation of nasal mucosa and skin.	Remarkable recovery of pulmonary function after oxygen treatment. Sinusitis improved by penicillin therapy.
45	1	0	B. coli B. aerogenes	Sore, reddened tongue and throat, increased cough.	Dyspnea and cough provoked by penicillin inhalations. Moderate relief from physiologic therapy.
46	1	Strep. viridans	Staph. aureus Non-hemolytic Strep.	0	Marked improvement on penicillin aerosol, sustained by one inhalation weekly for past 5 months.
47	1	0	B. aerogenes	0	Dyspnea and broncho-spasm became worse on penicillin inhalations, with slight relief from physiologic therapy.
48	1	Hemolytic Strep. Strep. viridans		0	Inhalations produced increased dyspnea. Chief benefit from oxygen.
49	1	Strep. viridans	B. coli Hemolytic. Strep.	Sore, reddened tongue.	Previously had marked improvement with physiologic therapy alone.
50	1	Pneumococcus, type 17 Staph. aureus	B. coli B. aerogenes	Coated brown tongue.	Striking improvement during hospital stay and for few weeks at home. Relapse not helped by occasional inhalations in clinic.
51	1	Staph. aureus Hemolytic Strep.	B. coli	0	Treatment of sinusitis with negative pressure and aerosol for two weeks. Temporary recurrence, but moderate improvement persists.

the inspiratory disc valve in the cylinder between the mask and the rebreathing bag is removed. A flow of 8 to 10 liters per minute of oxygen results in continuous nebulization of the drug, with a loss of the amount of penicillin nebulized in expiration. When this method is used a concentration of 10,000 to 20,000 units per cc. may be employed, 100,000 units then being given as a dose 5 to 7 times daily. In some cases an injection of 200,000 to 300,000 units of crystalline penicillin intramuscularly is given after the last dose at night to provide an additional blood level during the evening. In other resistant cases an intramuscular injection of 300,000 units of beeswax penicillin may also be given once a day for a preliminary period of 4 to 5 days until the infection has been controlled. Since penicillin blood levels have been demonstrated to be sharply lower during nasal respiration of the aerosol (previous studies from this clinic), patients should be instructed to breathe through the mouth also during mask administration of nebulized solutions. Turbulence in the nasal passages results in a high deposition of the drug on the mucous membrane of the nasal and oral pharynx.

A demand valve apparatus for automatic nebulization of penicillin in inspiration is now being used more extensively.*[41]

A more recent method is the use of the closed head tent for semi-continuous inhalation of oxygen and penicillin aerosol, as illustrated in the accompanying photograph (Fig. 1). The closed head tent has been recently used for oxygen administration with an injector attached to the regulator to deliver concentrations of 40 to 60 per cent.** When this apparatus is employed a flow of 10 liters per minute through the nebulizer is used to wash out the carbon dioxide given off by the patient. An additional precaution may be necessary, namely, opening the tent for a distance of one inch at the back of the apparatus by moving the canopy one inch forward. A solution of 10 cc. of penicillin containing 10,000 units per cc. is instilled into the nebulizer, either periodically or by a slow infusion drip. One hundred thousand units may be given in this way 5 to 10 times daily. In severe cases a total of 1,000,000 units of penicillin has been employed each 24 hours. At the conclusion of nebulization of penicillin it is desirable to rinse the nebulizer with normal saline and in cases of either spasm or swelling of the mucous membrane with 0.5 to 1 cc. of 1 per cent neosynephrine. Rinsing results in taking up the peni-

*The demand valve nebulizer devised by John H. Emerson Co., 22 Cottage Park Ave., Cambridge, Mass., is simpler and more satisfactory for this purpose than the one originally produced in our laboratory.

**Barach, A. L., Levenson, E., and Rumsey, C. C.: "The Use of an Injector Meter for Maintenance of a Prescribed Oxygen Concentration and Elimination of Carbon Dioxide in Closed Head Tents," *Am. J. Med.*, April 1947 (In Press).

cillin that is otherwise deposited in the nebulizer, in that way preventing clogging and also undue concentration of the drug.

A metal water bottle attached to the regulator also prevents undue concentration of penicillin in the nebulizer and results in a relatively higher humidity of the inhaled aerosol. When the oral reinhalation apparatus is used, the bag is filled with a glass of very hot water from the tap and the bag itself rests in a container that holds hot water. The water vapor coming from the bag is mixed with the aerosol, which makes for more comfortable as well as more effective therapy. Instead of the water bottle 0.5 cc. of saline may be instilled two or three times after treatment to dissolve concentrated penicillin in the nebulizer.

In the treatment of sinusitis a venturi valve was constructed that results in a negative pressure produced in the sinuses. After the release of the negative pressure, which is followed either by atmospheric pressure or by a slight positive pressure, the previously inhaled penicillin is introduced into the sinuses.* Crystal-

*The penicillin sinus apparatus may be obtained from the Inhalational Therapy Co., 4 East 41st Street, New York City; the Oxygen Equipment Mfg. Co., 405 East 62nd Street, New York City, and the Vaponefrin Co., 6812 Market Street, Upper Darby, Pa. The valve has been dispensed with in the present method of using only the venturi for production of negative pressure.

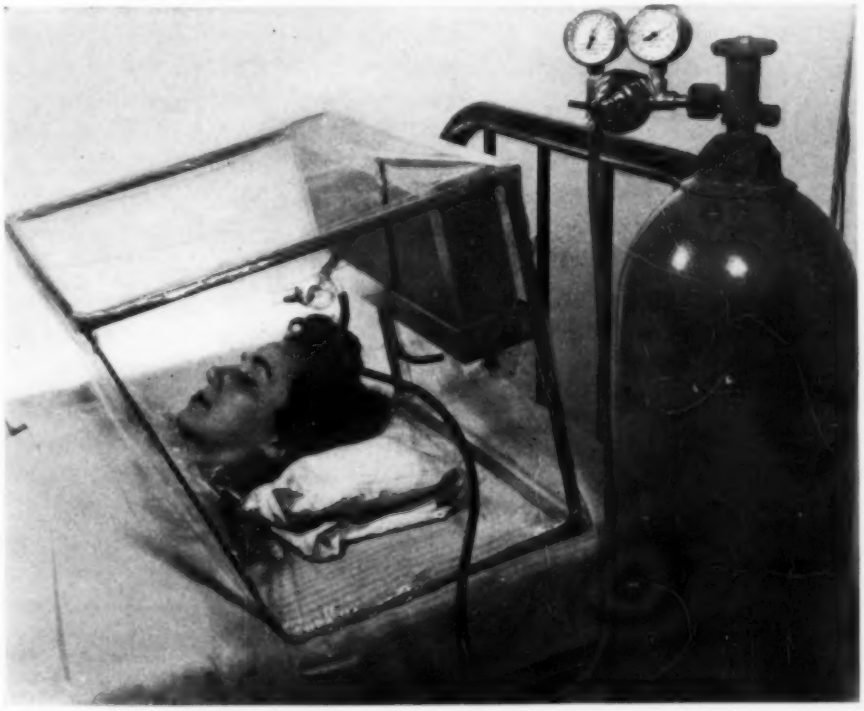

Figure 1: Head tent with closed top and nebulizer for penicillin aerosol and oxygen administration.

line penicillin is employed in sinus aerosol therapy at a concentration of 50,000 units per cc. The treatment is followed by a rinse of 0.5 cc. of 1 per cent neosynephrine and later by 0.5 to 1 cc. of normal saline. In acute cases that are handled in the office, 100,000 units appear to be preferable to 50,000 units, when the therapy consists of a single daily treatment. In cases that are treated at home, 50,000 units in 1 cc. of saline, are employed 4 times a day, in conjunction with repeated negative pressure. In cases of bronchopulmonary disease and sinusitis, additional aerosol inhalations may be carried out with oral inhalation of penicillin either by mask or with the mouthpiece method.

When streptomycin is used it may be combined with penicillin 0.5 grams being dissolved in 10 cc. of normal saline, together with 100,000 units of crystalline penicillin. When 2 grams of streptomycin are used in a 24 hour period, the mask nebulizing apparatus is a more comfortable, although less effective, method than the Y tube oral reinhalation apparatus. The frequency of fatigue when large quantities of solution are nebulized with the Y tube generally makes it preferable to use the mask nebulizing apparatus, or the automatic demand valve apparatus.

If sodium sulfathiazole or sodium sulfadiazine is employed, the rebreathing bag is removed, since the carbon dioxide given off by the patient will lower the pH of the solution and result in precipitation of sulfadiazine in the nebulizer. Other antibiotic and chemotherapeutic aerosols may be employed with the methods described above. The effectiveness of penicillin aerosol therapy is revealed by the blood level of penicillin measured from one-half hour to two hours after a treatment. In addition the concentration of penicillin in the expectoration after treatment provides a reliable indication of the topical application of the drug. In a few cases of pulmonary emphysema, bronchial asthma and pulmonary fibrosis penicillin aerosol produces an apparent swelling of the mucous membrane of the bronchi that might result in increasing dyspnea. This may either be the result of an allergic reaction to the drug or to a non-specific irritant effect. When dyspnea does take place following repeated inhalations of penicillin aerosol this method should be terminated. When the patient has recovered from a reaction of this kind, intramuscular injection of penicillin may be employed without necessarily causing a recurrence of the former reaction.

An attempt was made in this study to differentiate the improvement obtained from penicillin and that derived from physiologic therapy, which included oxygen and the measures described above. In most instances these patients had been previously benefited by this type of therapy with the result that the clinical improve-

ment listed under this heading in Table 1 will appear to be less important as a therapeutic procedure than it actually was. Although the previous course of physiologically directed therapy in some cases served as a baseline for control of penicillin treatment, continuation of both programs made difficult the appraisal of the importance of each. Our intention, however, has been to allocate as far as possible the varying roles played by physiologic and antibiotic therapy in the cases subjected to individual study.

Of 86 courses of treatment in 51 patients clinical improvement that appeared to be chiefly attributable to penicillin therapy was marked in 20, moderate in 19, slight in 25 and absent in 22. In the same series of cases improvement related to physiologic therapy was marked in 9, moderate in 36, slight in 23 and absent in 18. The response of the individual patient will be seen in Table 1. Of the total of 86 courses, 66 showed moderate or marked improvement. In analyzing the factor responsible for benefit, physiologic therapy appeared to play a more important role in 31 and penicillin treatment in 27. In 8 of these improved cases equal benefit was ascribed to penicillin and physiologic therapy.

In those patients who were grouped as being either moderately or markedly improved, the duration of improvement was considered temporary in 33 and longer than two months in 33. The patients who relapsed were in some cases treated with additional antibiotic therapy. Continuity of benefit in a few patients, especially in those with bronchiectasis and in occasional cases of pulmonary fibrosis, appeared to require a maintenance dose of penicillin. Under these circumstances inhalation of an aerosol of penicillin appeared more feasible.

In Table 2 the effect of penicillin aerosol on the organisms recovered from the sputum before and after treatment is shown. In the majority of cases the gram positive organisms disappeared from the sputum and instead gram negative organisms, especially B. coli or coliform organisms, B. aerogenes, B. pyocyaneus were found. This sequence of events was noted in our original report[22] and has been commented upon in subsequent studies.[35,36] In most instances it would appear likely that gram negative organisms that formerly were unable to grow out on a plate covered by gram positive bacteria now make their appearance as a profuse growth. In perhaps a few instances mixed infection with gram positive and gram negative bacteria may take place in chronic pulmonary disease. The favorable results reported by Olsen when penicillin and streptomycin aerosols were administered together in cases of bronchiectasis[36] would tend to support this concept.

However, since Abraham and Chain,[37] Woodruff and Foster,[38] and Meleney[39] have demonstrated that gram negative organisms

may produce an enzyme which nullifies the bacteriostatic effect of penicillin, the value of streptomycin may have been due in some instances to the fact that it destroys organisms that are not in themselves pathogenic, but are capable of elaborating penicillinase. In our experience patients with various types of bronchopulmonary disease, including bronchiectasis and lung abscess, have been at times markedly benefited by penicillin aerosol, even during the continued presence of gram negative organisms. In most cases in which sputum cultures are made a week or two following the termination of treatment, gram positive organisms again make their appearance and gram negative bacteria are found either in very small numbers or not at all.

Urticaria which at times takes place with administration of penicillin either by injection, by mouth, or by inhalation may now be handled by pyribenzamine or benadryl, or vitamin K.[40] In cases in which the primary condition is pulmonary emphysema, pyribenzamine, 50 mgm. five times daily, may be followed by significant improvement in bronchial spasm.

Another unfavorable reaction is the infrequent development of increased dyspnea due to an allergic reaction to penicillin aerosol. Although crystalline penicillin is now uniformly used when penicillin is given by inhalation, patients at times appear to develop a swelling of the bronchial mucosa apparently due to an irritant or allergic response to penicillin particles lodging in the respiratory passageway. Discontinuance of this form of therapy, with demerol and at times oxygen therapy, for four or five days have been employed when necessary. Subsequent administration of penicillin by intramuscular injection or by mouth has then been used without untoward results.

The comparative value of penicillin when administered as an aerosol and by systemic injection requires further study but a discussion of the factors involved and the available evidence has been presented elsewhere.[31-34] In patients who manifest bronchospasm, inhalation of a dense nebulized mist may produce increased wheezing. For this reason, intramuscular injection of the crystalline salt in high dosages has been frequently employed. For ambulatory patients two injections of 300,000 to 400,000 units in 5 cc. of saline may be used. Aerosol therapy in allergic patients is reserved for those who show resistant organisms which require a high local (sputum) concentration for their elimination.

Difficulties are inevitably encountered in appraising the value of a new therapeutic procedure. In the treatment of pulmonary emphysema the mechanical difficulty of breathing is frequently paramount, at times in the absence of demonstrable infection in the bronchi. In cases without purulent or mucopurulent expectoration antibiotic therapy would not appear to be indicated.

However, since bronchial obstruction is at times due to inflammatory swelling of the mucous membrane associated with active bronchial or bronchiolar infection, effective treatment with penicillin is of value by decreasing bronchial constriction. As in cases with bronchiectasis the duration of improvement may be substantial, the infection being controlled by repeated courses of therapy at intervals of one to several months. In other instances recurrence of infection takes place more promptly. Further studies are required to appraise the ultimate value of this form of treatment. That favorable results have occurred in many patients, as have been noted in this study and in the reports of Segal,[35] indicates that additional trial of antibiotic therapy, together with physiologically directed therapy, may improve the situation of the patient with pulmonary emphysema either in association with bronchial asthma or pulmonary fibrosis.

SUMMARY

A report is presented of the results of physiologic therapy and antibiotic (penicillin) treatment in patients with pulmonary emphysema. In this group of cases the associated diagnosis was more commonly bronchial asthma, less frequently pulmonary fibrosis, bronchiectasis, sinusitis and chronic bronchitis.

The results of treatment may be summarized as follows: Of 86 courses of treatment in 51 patients clinical improvement attributable chiefly to penicillin therapy was marked in 20, moderate in 19, slight in 25; in 22 no improvement took place. The results of physiologically directed therapy in the same group were marked improvement in 9, moderate in 36, slight in 23 and none in 18. Of a total of 86 courses, 66 cases manifested moderate or marked improvement. In analyzing the role played by each therapeutic factor as well as could be determined, 31 of the 66 improved cases appeared to be more influenced by physiologic therapy, 27 by penicillin treatment, with an equal effectiveness in 8 cases. In some of the cases treated in this series previous exposure to physiologic therapy had resulted in a baseline of improvement, which gave the appearance of less striking effect from this type of therapy than might have been anticipated in patients who had not been treated by the procedures used in bronchial relaxation and inhalational therapy. The study of the sputum culture before and after treatment revealed the characteristic disappearance of gram positive organisms in the majority of cases with replacement by gram negative bacteria.

In most cases penicillin was administered as an aerosol but favorable results were also obtained with systemic administration. The response to varying modes of treatment of the individual case is presented in tabular form.

RESUMEN

Se presenta un informe relativo a los resultados de la terapia fisiológica y del tratamiento antibiótico (penicilina) de enfermos con enfisema pulmonar. En este grupo de casos el diagnóstico asociado fue más comúnmente el asma bronquial, y menos frecuentemente fibrosis pulmonar, bronquiectasia, senositis y bronquitis crónica.

Pueden resumirse los resultados del tratamiento de la manera siguiente: De 86 series terapéuticas en 51 enfermos, en 20 se obtuvo mejoría clínica definida, imputable principalmente al tratamiento con penicilina; en 19 fue moderada, ligera en 25, y en 22 no tuvo lugar mejoría alguna. En el mismo grupo los resultados de la terapia fisiológica fueron: mejoría definida en 9, moderada en 36, ligera en 23 y ninguna mejoría en 18. De un total de 86 series, 66 casos manifestaron mejoría moderada o definida. Analizando, tan bien como pudo determinarse, el papel desempeñado por cada factor terapéutico, 31 de los 66 casos mejorados parecían haber sido más beneficiados por la terapia fisiológica, y 27 por el tratamiento con penicilina, con una eficacia igual en 8 casos. En algunos de los casos tratados en esta serie la previa exposición a la terapia fisiológica había producido una mejoría menos notable de lo que se habría podido anticipar en enfermos que no habían sido tratados mediante los procedimientos usados en la dilatación bronquial y la terapia de inhalación. El estudio del cultivo del esputo, antes y después del tratamiento, reveló en la mayoría de los casos la desaparición característica de los microbios Gram positivos y su reemplazo por bacterias Gram negativas.

En la mayor parte de los casos se administró la penicilina en forma de aerosol, pero también se obtuvieron resultados favorables mediante otros métodos de administración. Se presenta en forma tabular la reacción de cada caso a los varios tratamientos.

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Treatment of Thoracic Empyema with Sodium Tetradecyl Sulfate 1:500 in Azochloramid 1:3300

E. H. TONOLLA, M.D., and O. C. BRANTIGAN, M.D.
Baltimore, Maryland

The Principles of treatment of thoracic empyema continue to remain: (1) Adequate drainage without production of pneumothorax. (2) Obliteration of the empyema cavity by lung expansion, and (3) Sterilization of the area. These principles may be more adequately obtained and empyema combatted with less chance of sequelae if the methods used in the treatment are directed against the mechanical obstacles encountered in the lungs and pleura and also directed to influence the dynamic phases of the inflammatory process.

The Mechanical obstacles which at times are difficult to overcome are:

1. An elastic mesial pleura. This tends to prevent dissociation of the two lungs thereby allowing mediastinal shift to occur which may produce anoxemia and embarrass the patient's heart.[4,5,7-10] This may be prevented by the selection of a proper operative procedure (closed drainage, to be described) and further benefited by having a fluid medium between the chest wall and lung, instead of allowing a pneumothorax to occur.[1]

2. Broncho-pleural fistula may produce an almost insurmountable obstacle. This condition is present much more frequently than commonly thought and is probably the greatest cause of serious empyema. Broncho-pleural fistula is best treated by aiding the mechanical phases of the inflammatory process with the production of a fibrinous exudate which closes the fistula and by fixation which limits the spread of infection. Sodium Tetradecyl Sulfate, 1:500, in Azochloramid, 1:3300, usually will accomplish this result. It is occasionally necessary to use suction together with tetradecyl sulfate solution to aid in lung re-expansion.

3. Lung re-expansion is also influenced by the tone of the bronchial musculature.[3] Where tone is increased, tension in the alveolar sacs becomes greater and the lung expands rapidly as is seen at times in therapeutic pneumothorax after pneumonolysis, and is typical in the asthmatic. With this type of lung, the empyema is cured promptly after drainage because the pleural space

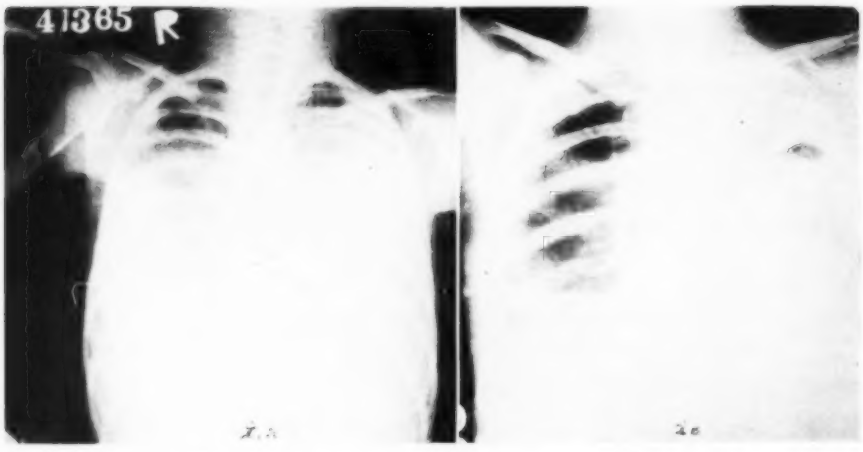

Figure 1-A

Figure 1-B

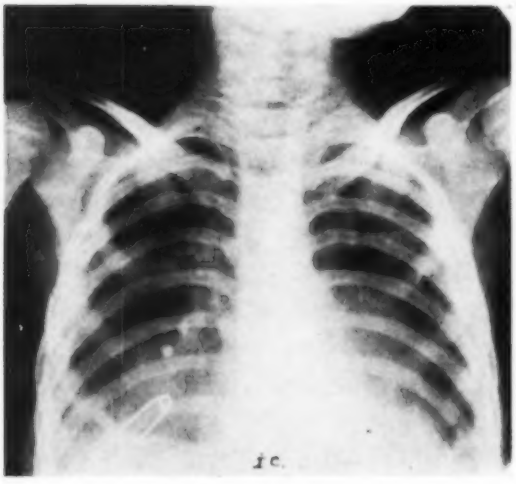

Figure 1-C

Fig. 1-A: Bilateral Empyema.

Fig. 1-B: Tube drainage (closed type) on Rt. Broncho pleural fistula, left.

Fig. 1-C: Bilateral tube drainage (closed type). Empyema cavities obliterated by lung expansion, without production of pneumothorax.

is quickly obliterated. With decreased tone or absence of bronchial tone, re-expansion of the lung is prevented and so cure is inhibited through delayed or incomplete obliteration of the empyema cavity. As long as a space with surfaces remain, sterilization is hindered. Delay in obliteration of the pleural space with continuation of infection produces a thickened pleura. The hypotensive lung may be combatted by the administration of drugs that will cause increased bronchial tone and, accordingly, increase intrapulmonary pressure.

The Action of Azochloramid 1:3300, Tetradecyl Sulfate 1:500 Solution: The phases of the inflammatory process represent a defense mechanism which tends to restore the part to normal function (John Hunter, 1728-1793, advanced this hypothesis). Some of these mechanisms are: Dilatation of the capillaries, escape of

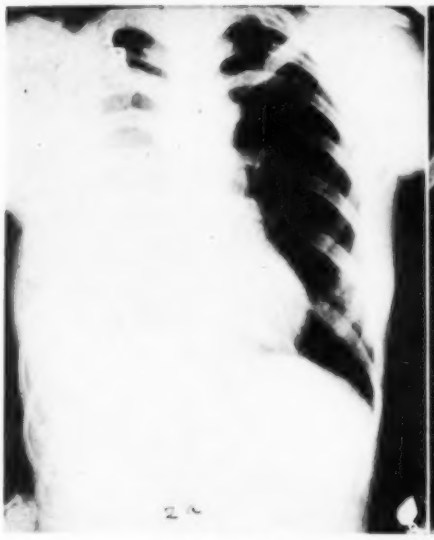

Figure 2-A

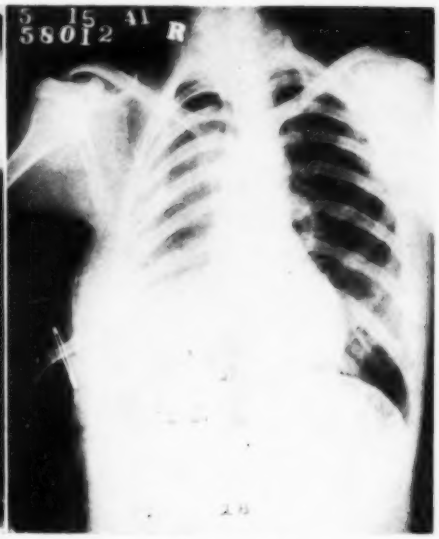

Figure 2-B

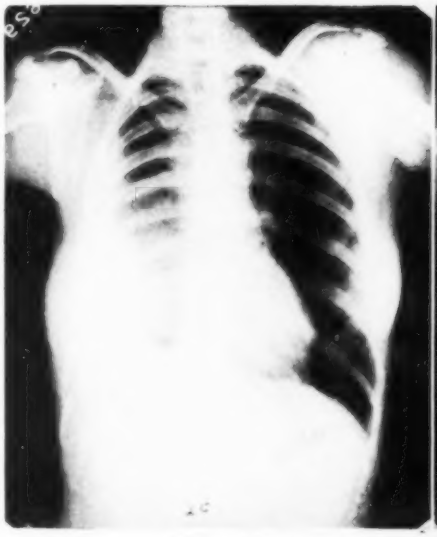

Figure 2-C

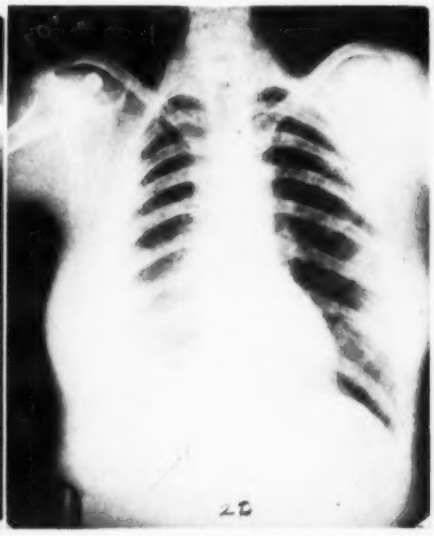

Figure 2-D

Fig. 2-A: Empyema, Right. — *Fig. 2-B:* Closed type tube drainage, with azochloramid sodium tetradecyl sulfate irrigations. — *Fig. 2-C:* Lung expanded. Soft fibrin in empyema cavity, remaining. — *Fig. 2-D:* Fibrin absorbing.

fluid from the vessels, diapedesis of phagocytes to the affected part and fixation which prevents the spread of infection or irritant by blood stream or lymphatics. A solution of sodium tetradecyl sulfate 1:500 in azochloramid 1:3300 alters the mechanical phases of the inflammatory process as follows: (1) It produces an exudate rich in fibrin which aids in the process of fixation, thereby prevents spread of infection and tends to hasten the closure of

bronchopleural fistula. (2) The solution stimulates diapedesis of phagocytes to the infected area, thereby aiding the sterilization of same. (3) The solution lowers surface tension of existing pleural exudate, allowing better penetration of the antiseptic solution and facilitates removal of the exudate by tube or needle. (4) Formation of pleural fibrosis is lessened. (5) The gelatinous fibrin which forms aids in the obliteration of the empyema cavity and in lung expansion without leaving behind a thickened pleura.

Occasionally after a period of use, azochloramid produces a febrile reaction. This is apparently a sensatization reaction against azochloramid and when it occurs, some other bacteriocidal agent can be used with sodium tetradecyl sulfate. Other bacteriocidal agents have been used in the treatment of empyema both with and without sodium tetradecyl sulfate but the azochloramid, sodium tetradecyl sulfate combination has been found to be the most effective. Without sodium tetradecyl sulfate the bronchopleural fistulae will not close so readily.

Technique of Treatment

1. *Non-Operative:*

Aspiration of the pleural cavity with insillation of antiseptics or chemotherapeutic agents, may suffice in healing an empyema.[2] If this method is used, it is important to have daily aspirations and instillations for the first three to six days or until the infection is evidently eliminated or adequately controlled. If the infection is not eliminated in three to six days, the method should be abandoned for a more successful manner of treatment, either closed or open thoracotomy. Once infection is eliminated, it is important to continue aspirations and instillations at longer intervals until the pleural space is completely obliterated. Carrying the aspirations and instillations to the point where complete obliteration of the pleural space is obtained, is the most difficult aspect of this method of treatment and often accounts for its failure. Healing of the parietal and visceral pleura finally cures the empyema in this or any other type of treatment. If fluid continues to form, then this method will fail. If air is permitted to remain in the pleural space after instillation, the method will fail. It is difficult to destroy the last bacteria if a space remains within the pleural surfaces, but if the remaining few bacteria are caught between two healing surfaces they will be eliminated.

2. *Closed Tube Drainage:*

The closed tube method of drainage is the easiest, simplest and most efficient method. It makes less work for the doctor with less

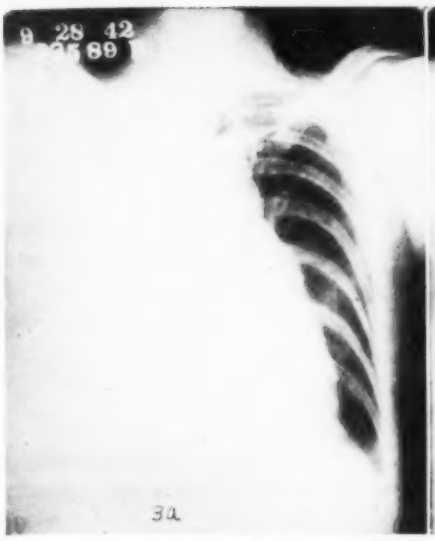

Figure 3-A

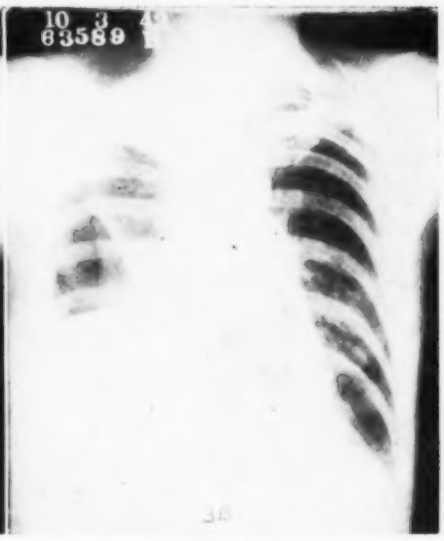

Figure 3-B

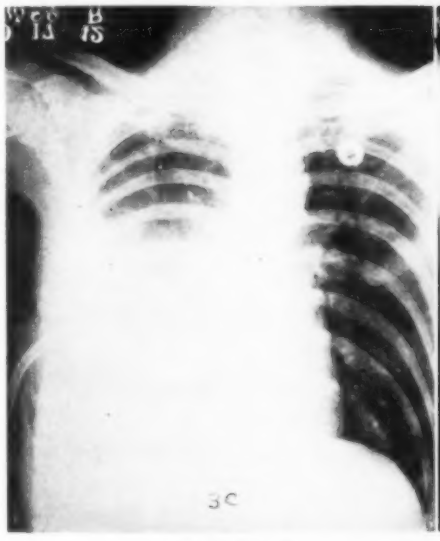

Figure 3-C

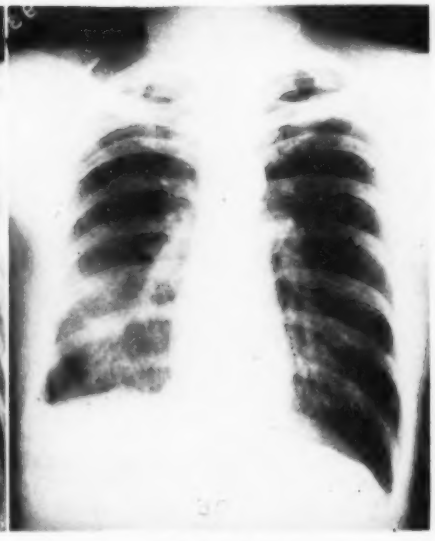

Figure 3-D

Fig. 3-A: Empyema, Right, with mediastinal shift to left. — *Fig. 3-B:* Bronchopleural fistula with pneumothorax present. — *Fig. 3-C:* Closed type tube drainage with azochloramid, sodium tetradecyl sulfate irrigations. — *Fig. 3-D:* End result. Re-expansion without functional disability.

discomfort to the patient, and this is certainly true when compared with open drainage. The site for the introduction of the tube can be determined by locating the pus with an aspirating needle. If the empyema is large or general the best site for introduction of the tube is in the 8th interspace in the posterior axillary line; however, the tube can be inserted at whatever location the pus

is found. The site selected for the introduction of the tube is anesthetized with procaine 0.5 per cent. A stab wound is made in the skin and then a trocar and cannula are thrust into the pus in the pleural cavity. The trocar is removed and a mushroom or straight catheter is inserted through the cannula which is then removed. The catheter should be practically the size of the cannula. The tube is anchored with adhesive. Introduction of air into the pleural cavity is guarded against during this maneuver. The intrapleural catheter is then connected to a "Y" or "T" tube with one limb leading to a trap bottle and *opening under water*. The other limb of the "Y" or "T" tube is connected to an overhanging flask containing the irrigating solution. Sodium tetradecyl sulfate, 1:500, in azochloramid, 1:3300, is preferred as the irrigating solution because of the properties above mentioned. The nurse is instructed to irrigate with 100 cc. or less of solution (depending upon the size of the pleural pocket) every four hours. The tube is left in the pleural cavity and the apparatus kept intact until the pleural space holds approximately one ounce. The intrapleural tube is then removed and a small silver wire is inserted in its place to act as a vent until the final pocket obliterates itself. If needed, a negative pressure suction can be applied to the trap bottle but this is rarely necessary.

Sulfanilamid 5 per cent in a vanishing cream base containing sodium tetradecyl sulfate 0.2 per cent is used about the tube as it emerges from the chest wall. It has been found that this prevents infection about the tube and tends to prevent air from entering the pleural cavity around the tube.

3. *Open Thoracotomy:*

The indications for open thoracotomy and contraindications to closed drainage are (1) where a questionable diagnosis exists; if one suspects an abscess of the lung, an infected cyst of the lung, or a tumor, etc., an open thoracotomy, perhaps, will lead to the correct diagnosis. (2) Where the empyema is located in the neighborhood of important structures that might be injured by the trocar or tube. This is especially true in small pockets against the mediastinum. In such situations the open operative attack is much the safest. (3) Where the pleura is markedly thickened before treatment is instituted. In such a patient complete healing is delayed with either method of treatment, but with open drainage the patient can be discharged from the hospital sooner and, therefore, it is often indicated instead of closed drainage.

It is important to point out that open thoracotomy, even though it is not preferred, is efficient on all types of empyema except,

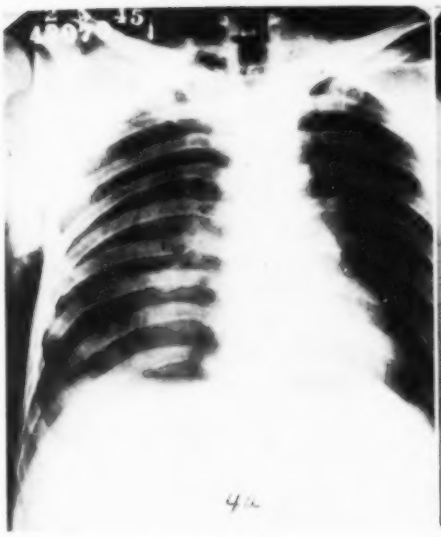

Figure 4-A

Figure 4-B

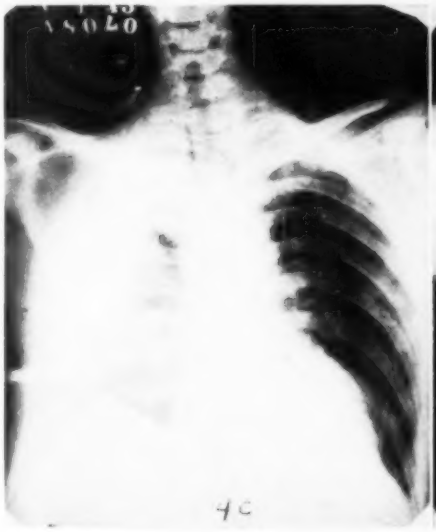

Figure 4-C

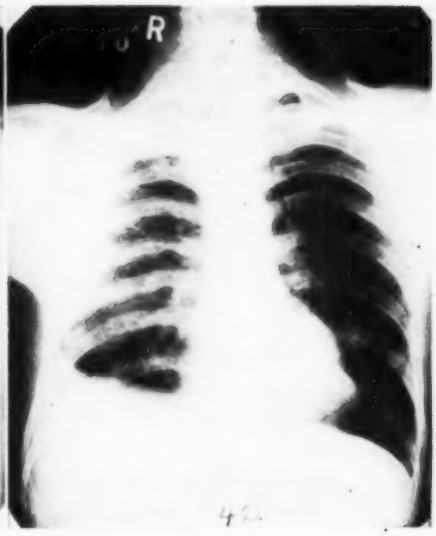

Figure 4-D

Fig. 4-A: Multiple fractured ribs, Right, with marked subcutaneous emphysema. Patient also had fractured pelvis and compound fracture of femur, tibia and fibula, Right. — *Fig. 4-B:* Pyo-hemo-pneumothorax with mediastinal shift to left and broncho-pleural fistula. — *Fig. 4-C:* Closed type tube drainage with suction to relieve mediastinal shift and irrigation with azochloramid sodium tetradecyl sulfate solution. — *Fig. 4-D:* End result. Re-expansion without functional disability.

(1) where there is no fixation of the lung and mediastinum, (2) where the patient's condition is critical, either because of concommitant disease or old age and (3) where there is a generalized empyema even though the mediastinum is fixed.

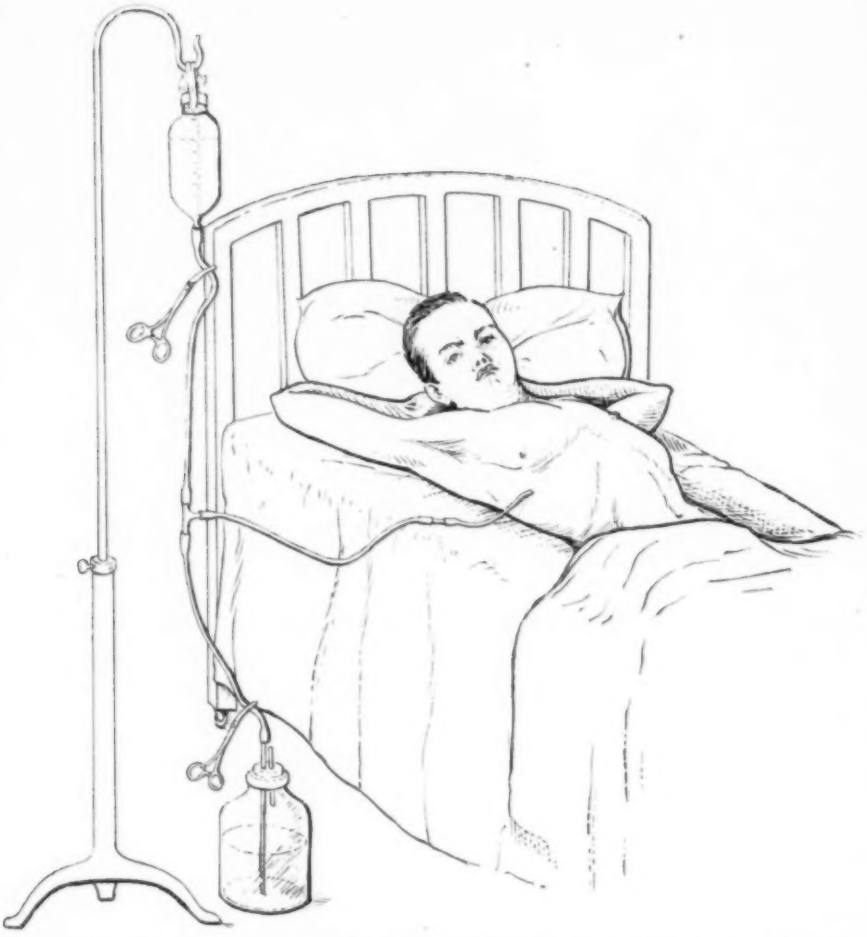

Figure 5: A simple method of tube drainage with apparatus for irrigating the pleural cavity. The irrigating fluid is usually allowed to remain in the pleural cavity for thirty minutes and the clamps are placed as the drawing indicates. At the end of this period the inferior clamp is removed and the pleural cavity is thus under siphonage drainage. When the pleural cavity is to be instilled the inferior clamp is applied and the superior clamp released until the desired quantity of fluid enters the pleural cavity and then the clamp is re-applied.

Results

Seven patients have been treated by aspiration and instillation. Six have recovered and remained well. One patient continued to have fever but seemed much improved. Open thoracotomy was done and the fever continued for three weeks. The source of the fever was never determined but obviously it was not from the pleural cavity. The patient recovered.

Twenty-eight patients were treated by closed drainage. A child, age 6 months, died three weeks after treatment was instituted. The cause of death is unknown. In two patients the tube was

removed inadvertently before the pleural space was obliterated. In one of these two the result was good but there resulted a thickened pleura and in the other open thoracotomy was resorted to. Finally the wound healed to a small opening and a Brewer tube was inserted and thus it was converted to a closed type of drainage with suction. This is the only case where the threat of a chronic empyema was eminent. It seems that in this case complete cure can be expected.

Twenty patients have been treated by open thoracotomy and all have made rather uneventful recoveries. In none has there been a chronic empyema.

CONCLUSIONS

1. No chemotherapeutic agent or combination of agents will replace adequate surgical procedure and technique in the treatment of empyema.

2. Sodium tetradecyl sulfate, 1:500, in azochloramid, 1:3300, seems to be the best chemotherapeutic agent available in the treatment of acute pyogenic empyema, especially if bronchopleural fistula or mediastinal shift is present.

3. Azochloramid tetradecyl solution lowers surface tension, thereby allowing better penetration of the antiseptic agent and facilitates removal of the exudate. In addition, azochloramid tetradecyl sulfate solution tends to prevent reformation of intrapleural fluid while sterilizing the area.

4. Close attention must be given to the mechanical aspect of the pleural cavity.

CONCLUSIONES

1. Ningún agente, o combinación de agentes quimioterapéuticos, puede reemplazar a procedimientos y técnicas quirúrgicas adecuadas en el tratamiento del empiema.

2. El sulfato tetradecílico de sodio, del 1:500, en azocloramida, del 1:3300, parece ser el mejor agente quimioterapéutico a nuestra disposición para el tratamiento del empiema piógeno agudo, especialmente si existe fístula broncopleural o desviación del mediastino.

3. La solución de azocloramida y sulfato tetradecílico disminuye la tensión superficial y, en esta forma, permite la mejor penetración del agente antiséptico y facilita la evacuación del exudado. Además, la solución de azocloramida y sulfato tetradecílico, tiende a evitar la reformación del líquido intrapleural al mismo tiempo que esteriliza la cavidad pleural.

4. Debe prestársele gran atención al aspecto mecánico de la cavidad pleural.

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Treatment of Tension Cavities with Pneumothorax*

I. D. BOBROWITZ, M.D., F.C.C.P.**
Otisville, New York

Various authors[1-5] from clinical, roentgen, contrast substance or necropsy studies have emphasized that the essential condition in the healing of tuberculous cavities consisted in the closure of the draining bronchi. Pinner[6] stated that bronchial occlusion might be one method of healing cavities, but that it was not the only possible mechanism. He showed an open bronchus running into the scar of a closed cavity.

It is agreed that cavity closure can occur with the draining bronchus closed or open but not when partial bronchial obstruction causes a valve mechanism. The valvular bronchial obstruction can be due to changes within the lumen of the bronchus (caseous material, tenacious secretion or mucopurulent exudate), the bronchial wall (mucosal edema, tuberculous inflammation, ulceration, or hyperplastic infiltration, caseous bronchitis, fibrostenosis, or peribronchial fibrosis), or extra bronchial conditions (lymph node pressure). Under these conditions, with inspiratory dilatation of the bronchi, air enters the cavity and with expiratory contraction of the bronchi less air leaves the cavity, entrapping air and increasing the cavity pressure and tension.

Pulmonary collapse with relaxation or retraction, or actual compression of the lung will fail to close a tension cavity, for the positive pressure prevents collapse of the cavity walls. Eloesser[7] has stated that cavities ballooned from within, resist collapse measures unless the anatomy of the communicating bronchus can be changed, which will either open it and release the intracavitary pressure, or close it entirely so that the air within the cavity can be absorbed.

There is general agreement among phthisiologists that tuberculous tension cavities are not successfully treated by pneumothorax. Surgical measures have been considered necessary although there has not been a unanimity of opinion about the most efficacious operative procedure. The use of a Monaldi type of drainage alone has not often resulted in permanent cavity closure. A combination

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**Medical Superintendent, Municipal Sanatorium, Otisville, N. Y. (one of the institutions of the Department of Hospitals, City of New York).

of transthoracic intracavitary suction drainage and thoracoplasty has been advocated by some,[8] while pulmonary resection has been the choice of Maier,[9] and the utilization of skin-flap drainage in conjunction with other collapse measures has also been tried.[10]

However, the closure of tension cavities by partial re-expansion of pneumothorax has been mentioned by Rafferty[11] and others. Several series of cavity closures with re-expansion have been reported. Brunn, et al[12] observed 14 cases where, in spite of an adequate pneumothorax collapse, the cavity remained open and with re-expansion of the lung the cavity promptly closed. They believe this was associated with a subsidence of an allergic inflammation in the bronchus.

Steele, et al[13] described 12 cases of unexpected closure of cavities following re-expansion after ineffectual pneumothorax (usually because of adhesions), though no conclusions were made as to the mechanism of closure.

Shipman[14] described a case of bilateral pneumothorax with open bilateral cavities where, on re-expansion of the lungs, the cavities closed. This is explained as a result of the draining bronchi opening up.

The bronchial factor in cavity closure is indicated in a report by Meyersburg, et al.[15] They describe 3 pneumothorax cases with a ball-valve type of bronchial obstruction proximal to cavities that remained open. After bronchial aspiration, a satisfactory pneumothorax was obtained in each patient. The dynamics of the cavity closure was not explained.

Slavin[16] would use positive pressure pneumothorax to compress tension cavities. He describes two cases successfully treated in this manner.

The purpose of this paper is to present illustrations of the closure of tension cavities with pneumothorax. Tension cavities comprise such a significant cause for failures in collapse therapy that any method that can achieve success in their treatment is important. We have also attempted to determine the modifications of the pneumothorax or the factors responsible for the closure. Our cases could be classified according to specific groups which will be described.

GROUP 1 — Closure Of A Tension Cavity By Considerable Re-expansion Followed By Increase Of Pneumothorax

Case 1, Group 1: Prior to the initiation of pneumothorax, the cavity in the left upper lobe (Fig. 1) had several characteristics of a tension type—its large size, thin wall and scant surrounding infiltration. With the pneumothorax well established (Fig. 2) a definite change in bronchocavitary drainage had taken place and the cavity now had evidence of tension or positive pressure. It was huge and stood out, uncollapsed

in the pneumothorax space, while the remainder of the lung was markedly collapsed. It was not held out by adhesions (an intrapleural pneumonolysis had been performed with severance of all adhesions) but was ballooned out by the intracavitary positive pressure. The cavity enlarged with pneumothorax to this huge size after it had been originally diminished by the collapse which showed the assumption of intracavitary tension. A fluid level appeared only after induction of pneumothorax and this indicated the development of some degree of bronchial obstruction with impaired drainage from the cavity. The production of this tension cavity could be ascribed to a partially obstructed bronchus that permitted more air to enter during inspiration than could leave with expiration and the retained air built up the pressure within the cavity. The pneumothorax by its collapse of the lung, therefore altered the dynamics of the bronchocavitary drainage and was instrumental in the creation of the valvular bronchial obstruction. The distention of the cavity seen in Figure 2 was enhanced because the intrapleural pressures were always subatmospheric after refills and this increased the

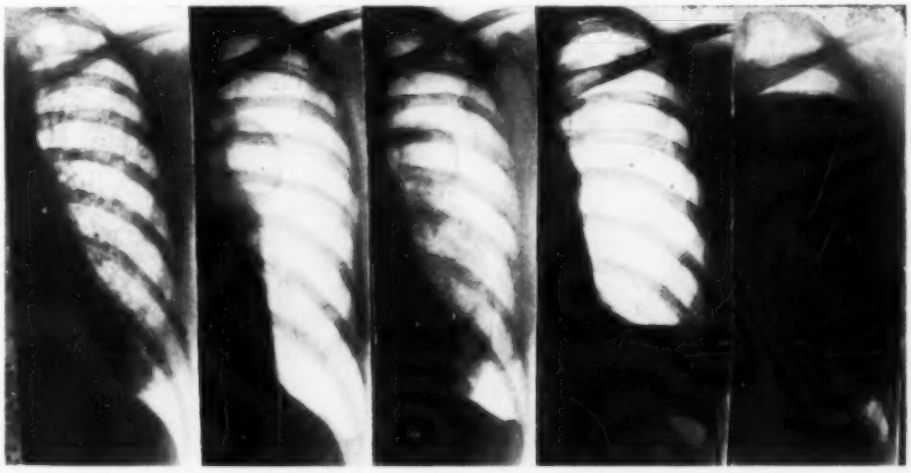

Fig. 1 Fig. 2 Fig. 3 Fig. 4 Fig. 5

Fig. 1, Case 1, 9/10/43: A.F., 25 year old white female. Admitted 9/9/43. Cavity in apical part of L.U.L., balloon shaped, 5 cm. x 4 cm., thin wall, scant surrounding infiltration. Nodular infiltration scattered throughout remainder of left lung and in 2nd and 3rd right anterior interspaces. Sputum highly positive.

Fig. 2, Case 1, 1/12/44: Left artificial pneumothorax induced 9/16/43; intrapleural pneumonolysis 12/18/43. Marked collapse of left lung with tension cavity 5 cm. x 4 cm., with fluid level in L.U.L. Small cavity, 1.3 cm. x 1.3 cm., below large cavity. Little fluid in costophrenic angle. Infiltration in right lung unchanged. Sputum persistently positive.

Fig. 3, Case 1, 2/10/44: Following discontinuation of refills for one month. Lung considerably re-expanded, cavity in L.U.L. smaller (3.8 cm. x 3.8 cm.) and more collapsed than remainder of lung. Small cavity below large vomica unchanged. Sputum positive.

Fig. 4, Case 1, 5/23/44: Marked collapse of left lung. No cavity visualized. Fluid below level of 10th posterior rib. Resolution in right lung with residual slight nodular fibrosis. Sputum negative.

Fig. 5, Case 1, 12/5/44: Marked collapse of L.U.L. maintained. Lower lobe re-expanded with lung margin at level of 9th posterior rib in axillary line. Fluid on level with 5th posterior rib. Chest tap revealed yellow, turbid, thin fluid, positive for tubercle bacilli on culture. Sputum negative.

difference between the positive intracavitary pressure and the negative intrapleural pressure.

The pneumothorax was obviously ineffective but surgery was not approved because stability of contralateral lesion was not certain. Re-expansion of the lung was decided upon with the possibility that cavity closure might occur with this procedure.

With discontinuation of pneumothorax for one month (1/11/44 to 2/11/44), sufficient re-expansion took place to alter the dynamics of the draining bronchus and change the intrabronchial valvular mechanism. Now (Fig. 3) without the addition of any intrapleural air, the lung expanded to a degree of 50 per cent collapse and the type of collapse showed interesting modifications. The cavity no longer protruded but showed more collapse than the remainder of the lung, indicating a diminution of tension or pressure within it. In other words, the collapse was becoming more selective and the cavity smaller. This result which occurred while refills were stopped, demonstrates that the attainment of a selective collapse with pneumothorax depends entirely on the changes within the bronchi and lung.

The marked changes in this case followed alteration of the bronchial drainage mechanism. Either the draining bronchus closed, permitting

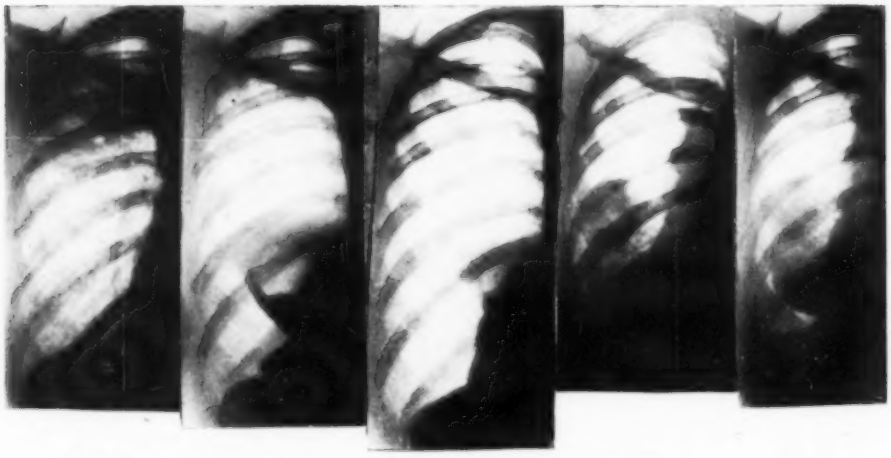

Fig. 6 Fig. 7 Fig. 8 Fig. 9 Fig. 10

Fig. 6, Case 2, 6/23/44: J.M.G., 23 year old colored female. Admitted 6/22/44. Large, thick walled cavity, 4 cm. x 4 cm., in lateral portion of R.U.L. Soft infiltration throughout R.U.L. Sputum highly positive.

Fig. 7, Case 2, 12/1/44: Right artificial pneumothorax induced 6/29/44. Refills stopped from 10/12/44 to 11/8/44 and then reinstituted. Right lung markedly collapsed and uniformly dense except for protruding, huge, tension cavity with fluid level. Inside of cavity 5 cm. x 4 cm. Sputum positive.

Fig. 8, Case 2, 1/31/45: Right lung about 85 per cent collapsed. R.U.L. sticks out very slightly. Lung airless except for cavity which is smaller, 2 cm. x 2 cm., without a fluid level. Sputum positive.

Fig. 9, Case 2, 6/27/45: After discontinuation of refills for 3 months. right lung collapsed about 60 per cent and no longer homogeneously dense, indicating increase in air content. Cavity in R.U.L., 2.5 cm. x 1.3 cm. and with apparent fluid level. Sputum positive. Fluid in pleural space at 11th rib posteriorly.

Fig. 10, Case 2, 9/29/45: Right lung maintained at 60 per cent collapse. Cavity for first time not visualized and replaced by dense homogeneous area. Sputum negative. X-ray picture unchanged for remainder of patient's stay in Sanatorium.

air in the cavity to be absorbed, or it more fully opened and this allowed the lung to relax and retract. However, one thing was certain—the check-valve mechanism in the draining bronchus no longer persisted. It was, therefore, now possible to continue pneumothorax effectively with the rapid closure of the cavity. Refills were re-instituted 2/11/44.

After one month of pneumothorax treatments following the re-expansion of the lung (or 7 weeks after re-expansion was started) the sputum was converted and a patient who had been persistently and highly positive for 7 months (from 9/12/43 to 3/7/44) became negative. Actually 3 months after pneumothorax was re-instituted the lung was markedly collapsed again (Fig. 4) and for the first time no cavity could be visualized on the roentgenogram. Refills were continued at less frequent intervals and re-expansion of the left lower lobe occurred, but a marked selective collapse of the upper lobe was maintained (Fig. 5). There were no further roentgenographic changes while the patient was in the institution.

She remained negative until her discharge (2/25/45) on 14 48-hr. sputum concentrates, 7 gastric concentrates, and 6 gastric cultures (except that 1 gastric culture was positive, 6/21/44).

The sputum has continued negative during a 10 months' follow-up period after discharge. Her general condition has remained excellent. A satisfactory pneumothorax has been maintained with a minimal amount of fluid in the left pleural cavity.

In this individual a totally ineffective and harmful pneumothorax with a huge tension cavity was converted into a satisfactory pneumothorax by alteration of the lung collapse, by considerable re-expansion and then re-institution of refills. Sputum conversion and cavity closure rapidly followed this procedure. A thoracoplasty or pneumonectomy, which had been considered but delayed because of the contralateral lesion, then became unnecessary.

GROUP 2-A — Closure Of A Tension Cavity By Considerable Re-expansion of Pneumothorax

Case 2, Group 2: The roentgenogram prior to pneumothorax (Fig. 6) revealed a cavity which, although of huge size, was not characteristic of a tension one. But with collapse therapy the cavity assumed the typical appearance of a positive pressure or tension type. As in Case 1, the pneumothorax must have altered the cavitary drainage mechanism and formed a valvular obstruction which produced the tension cavity. Corroborative evidence of this was furnished by a bronchoscopy done 10/21/44. (Dr. M. S .Lloyd): "The mucous membrane throughout the right side is doughy and cyanotic, particularly in the lower lobe, and the branches of all the lobes are markedly reduced in diameter. The lower lobe and middle lobe contain stagnant, frothy secretion. The upper lobe is completely closed by a mucopurulent collection without air. The mucous membrane bleeds easily on being touched. On inspiration this bronchus can be seen to open and it closes at the beginning of expiration producing a check-valve mechanism."

An attempt was made to remove this check-valve by re-expansion of the lung. The method successful in Case 1 was tried and refills were stopped for one month (10/12/44 to 11/8/44). The lung re-expanded and showed a 50 per cent collapse and then refills were again resumed. However, unlike Case 1, the cavity did not close. In fact, after one month

of pneumothorax, following the partial lung re-expansion, the cavity (Fig. 7) was larger than it had ever been. A lobectomy was considered at this time but the patient refused surgery. The huge cavity stood out, uncollapsed, with the remainder of the lung markedly collapsed, indicating a positive pressure and tension within it. Because the intrapleural pressures had always been negative, the cavity would tend to balloon out more. Either the lung had not been re-expanded sufficiently or kept out with a partial collapse for a long enough period to produce the necessary changes in bronchial drainage. However, with continuation of pneumothorax, the lung collapse could be increased and the cavity became smaller (Fig. 8).

This change in the roentgen picture indicated that the check-valve mechanism or partial bronchial obstruction had been altered, for while the pneumothorax had been maintained with negative pressures, the collapse of the right lung was considerably changed. The right upper lobe was collapsed more and did not protrude as much as before and the cavity was much smaller and was no longer ballooned out because the tension or positive pressure was diminished.

But as the cavity persisted, it was decided to permit the lung to be re-expanded for a longer period of time. For 3½ months, from 3/27/45 to 7/10/45, no refills were given. Changes were rapid after this re-expansion period. On 7/17/45 the sputum was last reported positive (having been persistently positive for 13 months) and it subsequently remained persistently negative till the patient left on 11/22/45. The negative reports consisted of 7 sputum concentrates, 3 gastric concentrates and 3 gastric cultures.

By 6/27/45 the lung had re-expanded and had a 60 per cent collapse and the cavity was considerably reduced (Fig. 9). Three months later (Fig. 10) with the pneumothorax maintained at this degree of collapse, the cavity was definitely closed and thereafter no longer visualized. Four months after discharge the condition was unchanged.

The re-expansion of the lung to a degree of 60 per cent collapse was apparently responsible for the closure of the cavity. The lung expansion must have altered the bronchial drainage from the cavity so that the bronchus either opened (permitting the cavity to retract) or it completely closed, causing the cavity to collapse. In either case the partial bronchial obstruction or check-valve, the cause of the positive pressure or tension cavity, no longer existed. With the expansion of the lung and increase in its air content, sufficient pulmonary tissue was available to fill in the space of the shrinking cavity.

Once the proper degree of lung collapse was determined, it was easy to maintain the pneumothorax at an effective and satisfactory level. It was fortunate that a good clinical result was obtained with pneumothorax in this case for thoracic surgery had been refused.

GROUP 2-B — Closure Of A Tension Cavity By Partial Re-expansion Of A Pneumothorax Limited Because of Adhesions

*Case 3, Group 2:** Prior to pneumothorax the cavity (Fig. 11), though not spherical in shape, had several characteristics of a tension type for it was very large, with a thin wall and fluid level. The fluid level indi-

*The author wishes to acknowledge sincere gratitude for the abstract and films in this case so kindly furnished by Kingston Avenue Hospital (service of Dr. Foster Murray).

cated interference with bronchial drainage. During the first few weeks that the pneumothorax collapse was slightly increasing, the cavity first diminished in size and then enlarged, showing the development of intracavitary tension. That pneumothorax could produce variations in bronchial obstruction and drainage was shown by transitoriness of the cavity fluid level while the treatments were given.

A conditioning factor in this case was the presence of adhesions (Fig. 12), for by holding the lung out, the adhesions could prevent the typical appearance of a tension cavity. An intrapleural pneumonolysis had been attempted but was unsuccessful. After 5 months of pneumothorax (Fig. 13), the right upper lobe was collapsed 75 per cent and the cavity, though smaller, persisted. Pneumothorax refills were stopped at this time in expectation of doing a thoracoplasty. However, one month after discontinuation of refills (Fig. 14), the right upper lobe re-expanded and was only 40 per cent collapsed, and the cavity closed and the sputum turned negative. Refills were reinstituted and the pneumothorax maintained.

The rapid closure of the cavity with re-expansion points to the fact that with the variation in lung collapse, some change in the draining

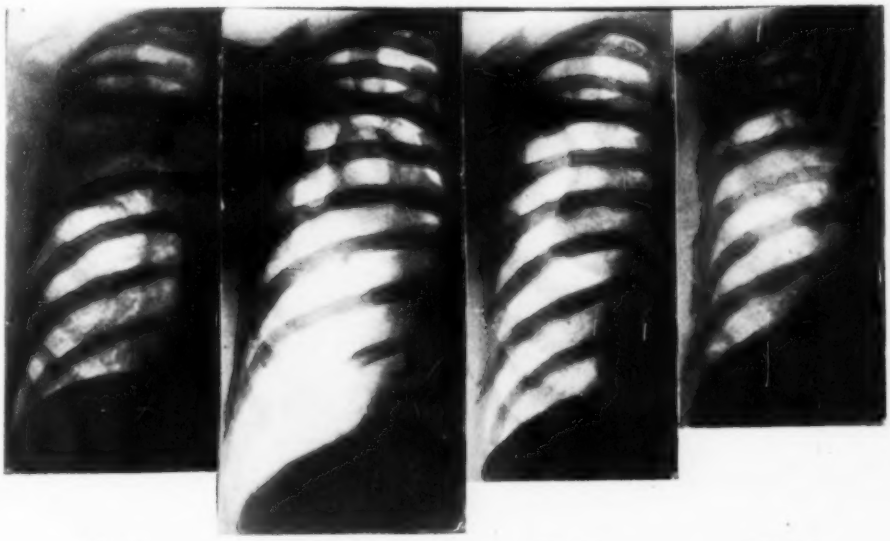

Fig. 11 Fig. 12 Fig. 13 Fig. 14.

Fig. 11, Case 3, 11/17/38: L.P., 33 year old colored male. Admitted to Kingston Avenue Hospital No. 1938. Cavity in R.U.L. between 1st and 2nd anterior ribs, 4 cm. x 4 cm., and with a fluid level. Soft mottled infiltration throughout R.U.L. except apex. Superior part of cavity wall visible and not thick. Sputum highly positive.

Fig. 12, Case 3, 1/27/39: Right artificial pneumothorax induced 11/22/38. R.U.L. about 50 per cent collapsed and diffusely airless except for large cavity, 3.5 cm. x 2.5 cm. No definite cavity fluid level. Several adhesion bands present. Lower lobes about 70 per cent collapsed. Sputum positive.

Fig. 13, Case 3, 4/26/39: R.U.L. collapse 75 per cent. Cavity smaller, 1.5 cm. x 1.5 cm. Lower lobes about 25 per cent collapsed. Sputum positive.

Fig. 14, Case 3, 5/26/39: One month after discontinuation of refills. R.U.L. about 40 per cent collapsed and well aerated. Cavity no longer seen and only scattered nodular and linear infiltration visible in its place. Lower lobes almost entirely re-expanded. Sputum negative.

bronchus must have occurred and the most plausible explanation would be the alleviation of a valvular or check-valve bronchial obstruction. In other words, the draining bronchus was either opened or closed and no longer partially obstructed and the cavity closed. Otherwise the cavity would have enlarged with re-expansion.

It may be argued that if the pneumothorax could have been further increased, the cavity would have closed as it had shown much diminution before re-expansion. But if more pneumothorax would have closed the cavity then with less pneumothorax on re-expansion the cavity should have become more apparent.

A phrenic crush was done on 8/23/39. After a few months a moderate effusion appeared in the right pleural space and the lung collapse was slightly increased.

Once the cavity was closed, the pneumothorax was easily maintained and it made no great difference if the collapse was then increased for there was apparently no check-valve bronchial mechanism to interfere.

The patient was in the Municipal Sanatorium, Otisville, N. Y., from 2/29/40 to 9/2/40. The chest picture remained about the same and all sputum examinations were negative.

During a follow-up period of 3½ years no cavities were seen and the sputum was negative.

GROUP 3 — Closure Of Tension Cavity Within Well Collapsed Lung By Considerable Re-expansion

In this group are considered pneumothoraces which are anatomically good but clinically ineffective. With re-expansion of the pneumothorax, a therapeutically satisfactory result is obtained.

Case 4, Group 3: Prior to pneumothorax the cavity in the right upper lobe (Fig. 15) had characteristics of a tension type, for it was large, spherical and with a thin wall (and slight surrounding infiltration). Several days after the initiation of pneumothorax (Fig. 16), the cavity had a fluid level, evidence that some degree of bronchial obstruction existed. After two months of pneumothorax therapy (Fig. 17) the right upper lobe was markedly collapsed and appeared homogeneously dense. For 10 months this marked collapse was maintained but the sputum was constantly positive. Re-expansion of the right upper lobe was attempted to obtain healing. Refills were stopped for one month (12/27/41 to 1/24/42) and the lung gradually re-expanded. The month that refills were stopped and the right upper lobe began to re-expand, a negative sputum was reported for the first time in a year. The second month after re-expansion was started the sputum definitely turned negative and remained so thereafter, and the third month the cavity closed (Fig. 18). During this period the right upper lobe was maintained in this same condition of re-expansion (at a degree of 50 per cent collapse). The conversion of the sputum and cavity closure was directly associated with the re-expansion and was no coincidental occurrence.

The patient was discharged on 11/8/42 as an arrested case with a daily work tolerance of 3 hours. The same lung collapse as illustrated in Fig. 18 was maintained and there were no other changes except that the round density or filled cavity slowly became smaller. Prior to discharge he had 11 negative sputum concentrates, 3 negative concentrates and 4 negative cultures. During a follow-up period of three and one-half years the patient has remained in excellent condition.

The lack of an endobronchial lesion (when a bronchoscopy was done all the visible bronchi showed no ulceration), and the persistently positive sputum pointed to an uncollapsed cavity in the right upper lobe. A cavity could reasonably persist in a markedly collapsed lobe only if a bronchial valvular obstruction existed that caused a tension or positive pressure within the cavity so that it could not collapse.

The fluid level in the cavity, before it was lost to view within the collapsed lung, indicated some bronchial obstruction. This obstruction could become valvular in nature, forming a typical check-valve mechanism. We have seen that pneumothorax, as in Cases 1 and 2, could be instrumental in altering bronchial drainage so as to produce marked tension cavities. The size and distensibility of a tension cavity will vary with many conditions—the degree of bronchial closure, accumulation of material in the cavity, thickness of the cavity wall, amount of infiltration, degree of elasticity, or presence of atelectasis in the surrounding lung, pressure in the intrapleural space and the presence of adhesions. Although a tension cavity is typically huge because of the marked positive pressure and considerable amount of air within the cavity and air accumulation with inspiratory phases of respiration, still it is possible to have a small tension cavity. In other words, the amount and entrapment of air and degree of positive pressure in the cavity can be slight or great. In Case 2, for example, a huge tension

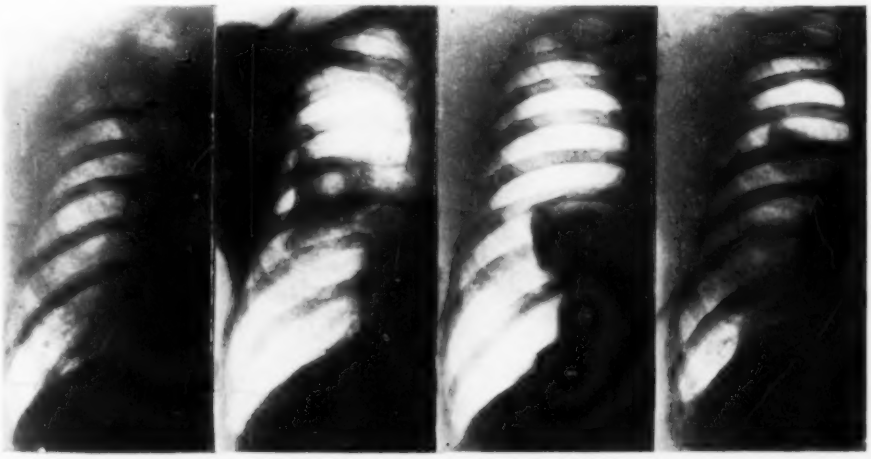

Fig. 15 Fig. 16 Fig. 17 Fig. 18

Fig. 15, Case 4, 2/21/41: W.W., 39 year old white male. Admitted 2/20/41. Thin walled cavity in right sub-apical area, 3 cm. x 3 cm. Slight mottling, medial and inferior to cavity. Few nodules in 2nd anterior interspace. Sputum highly positive.

Fig. 16, Case 4, 3/13/41: Right artificial pneumothorax induced 3/10/41. R.U.L. about 50 per cent collapsed. Cavity 3 cm. x 2½ cm., with a definite fluid level. Rest of lobe diffusely dense. Marginal collapse of lower lobe. Minimal fluid in costophrenic sinus. Sputum positive.

Fig. 17, Case 4, 5/19/41: R.U.L. about 80 per cent collapsed and homogenously dense. No cavity visible. No adhesions. Lower lobes about 70 per cent collapsed. Sputum highly positive.

Fig. 18, Case 4, 3/19/42: Right lung considerably re-expanded. Marginal collapse of lower lobes. R.U.L. about 50 per cent collapsed. No cavity visible. In its place a round homogeneously dense shadow undoubtedly filled-in cavity seen. Slight mottling medial to this round focus. Sputum negative.

cavity became much smaller with an increase of pneumothorax collapse, yet evidence of bronchial obstruction and intracavitary tension persisted. In this patient, Case 4, soon after pneumothorax was initiated, the appearance of the fluid level showed the cavity partly filled so that with an increased collapse, a good part of the cavity could be filled and only a small cavitary space might remain and persist because of a check-valve bronchus. Eloesser[7] found from manometric studies in post mortem lung cavities that even irregular, ragged walled cavities in densely infiltrated, largely airless lung, contain air which is under more than atmospheric pressure much of the time. He stated that cavities may not appear to be blocked or ballooned yet the bronchi are not freely open and secretions, pus or necrotic bits of material block them partially part of the time and inflation results by entrapped air.

Although the cavity in this patient could not be seen within the markedly collapsed lung, I believe it existed within the collapsed right upper lobe as a small tension cavity hidden by the dense surrounding lung. The rapid conversion of the sputum with re-expansion is explainable on the basis of cavity closure resulting from modifications in the draining bronchus. As the lung re-expands, there is an alteration in the direction of the bronchus and a change in the size of the bronchial lumen and variation in bronchocavitary drainage. As has been emphasized, a cavity can close only if the draining bronchus is open or closed, but not check-valve in nature. With re-expansion the valvular obstruction must have been relieved and the bronchus was either opened or closed.

There were 2 other patients observed similar to Case 4. In both individuals, white male adults, thin-walled, right apical cavities were present, which were spherical in shape and measured 2 cm. in diameter. In each instance there was practically no surrounding infiltration. The sputum was positive and pneumothorax was initiated.

The pneumothorax was unsatisfactory and both patients required intrapleural pneumonolyses in order to sever apical adhesions and entirely free the upper lobe. The degree and type of collapse obtained after operation was similar in each case. The right upper lobe was about 90 per cent collapsed with no cavity visible within the lung. The lower lobes were collapsed 80 per cent in one instance and 50 per cent in the other. The pneumothorax was continued with the upper lobes 90 per cent collapsed. The sputum remained highly and persistently positive in spite of the excellent collapse. Bronchoscopies failed to reveal endobronchial tuberculous ulceration in either case. One patient remained positive for 9 months under these conditions and the other was positive for 13 months (but only 9 months with the upper lobe 90 per cent collapsed).

Re-expansion of the lung resulted in a rapid conversion of the sputum. In one case, within one month after re-expansion was started and with the right upper lobe 50 per cent collapsed, the sputum turned negative. The right upper lobe was allowed to expand to a degree of 25 per cent collapse and maintained that way with the patient constantly negative. At the site of the original cavity only an area of irregular fibrosis was seen. This patient, after sputum conversion, remained persistently negative for 11 months in the sanatorium on 13 sputum concentrates, 10

gastric concentrates and 10 gastric cultures. He was discharged as an arrested case with a work tolerance of 4 hours daily.

In the second patient, within 6 weeks after re-expansion was started, with the right upper lobe 60 per cent collapsed, sputum conversion occurred. No cavity was visible and only a coalescent nodular lesion remained in the right upper lobe. This person remained negative for 6 months on 11 sputum concentrates and 3 gastric concentrates. He was discharged as an arrested case, with a daily work allowance of 2 hours.

These patients are very similar to Case 4. A marked (90 per cent) collapse of the right upper lobe was present for many months, yet the sputum remained persistently positive. No endobronchial lesion was observed. These findings pointed to the presence of an uncollapsed cavity in the markedly collapsed lobes as the source of the sputum. With re-expansion of the lung, sputum conversion occurred rapidly (within 4 weeks and 6 weeks respectively) and no cavities were seen in the re-expanded lobes. The explanation offered is the same as for Case 4—that the cavities persisted in the markedly collapsed lobes as small tension cavities because of valvular bronchial obstruction, and with re-expansion the valvular obstruction was relieved, permitting rapid closure of the cavities and sputum conversion.

An anatomically satisfactory pneumothorax is usually considered a requisite for a proper clinical result. The degree of collapse may be progressively increased with the expectation that a good or marked collapse will be therapeutically effective. The experience in this group indicates that this is not necessarily so. A marked pneumothorax may be ineffective because of a hidden tension cavity within the collapsed lung. In these cases, in spite of an adequate collapse and lack of adhesions, if the sputum is not converted in a few months, re-expansion of the pneumothorax should be considered.

GROUP 4 — Closure Of Tension Cavity With Small Pneumothorax Collapse

Case 5, Group 4: The roentgen picture before pneumothorax was quite typical of a check-valve or tension cavity (Fig. 19). The cavity was very large, almost spherical in shape, with a thin wall and little surrounding infiltration and suggestive fluid level.

After pneumothorax was initiated there was further evidence of changes in the cavity drainage mechanism. There were transitory appearances of the intracavitary fluid level which indicated alterations in the lumen of the draining bronchus. The cavity decreased in size and then temporarily enlarged, even though there was no change in the amount of pneumothorax space, showing variations in the intracavitary pressure, probably secondary to bronchial changes.

The huge cavity, when closed, formed a very small, filled focus, indicating that the cavity had been under tension and there had actually been little lung destruction.

In this case there was rapid diminution in the size of the cavity with closure four months after initiation of pneumothorax (Figs. 20 and 21). Since then to date (four months after cavity closure), there has not been much change on roentgen examination. The closed cavity has become smaller. The sputum has been negative on 15 sputum concentrates, 4 gastric concentrates and 2 cultures.

This cavity closure took place in the presence of a small and anatom-

ically unsatisfactory collapse. This points to the closure mechanism being related principally to changes in the draining bronchus. This is also indicated by the fact that the cavity decreased in size and closed while the same degree of lung collapse was maintained. Apparently the partial pneumothorax was sufficient to alter the draining bronchus in such manner as to modify the bronchial check-valve and cause closure of the bronchus. With this the air within the tension cavity absorbed and the cavity collapsed and closed. The space left by the shrinking cavity was filled in by normal or emphysematous lung. Moreover, the adherent lung would prevent relaxation and retraction of the lung necessary to obtain closure with an open bronchus, and this would corroborate the idea of bronchial occlusion.

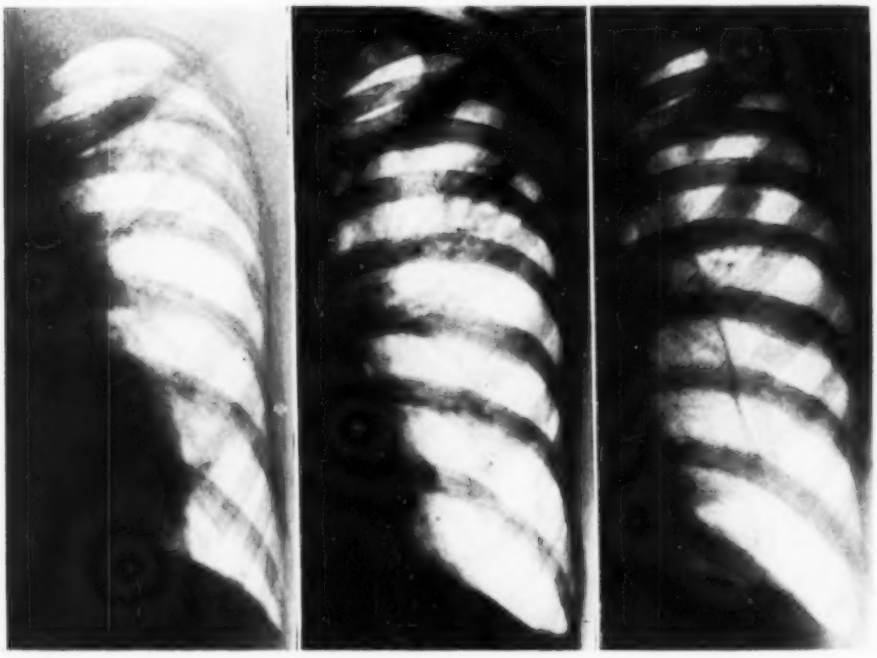

Fig. 19 Fig. 20 Fig. 21

Fig. 19, Case 5, 12/15/44: R.E., 22 year old white male. Admitted 12/14/44. Thin walled cavity in L.U.L., 4.2 cm. x 3.7 cm., with suggestive fluid level and little surrounding infiltration. Bronchocavity junction widened at inferior medial angle of cavity. Slight nodular infiltration scattered in 1st and 2nd anterior interspaces. Scattered soft coalescent lesions on right between 2nd and 5th anterior ribs. Sputum highly positive.

Fig. 20, Case 5, 1/31/45: Left pneumothorax induced 1/23/45. L.U.L. adherent to chest wall to level of 3rd anterior rib. Cavity smaller, 3 cm. x 2.7 cm., with a faint fluid level. Narrow peripheral collapse of rest of left lung. Air in front and around lung as some seen forming a pocket along upper mediastinum, collapsing inner or medial portion of lung slightly. Fluid fills costophrenic sinus. Sputum positive.

Fig. 21, Case 5, 5/31/45: Collapse of L.U.L. increased to about 30 per cent but it is adherent between 1st and 3rd ribs with air present in inner apical region. L.L.L. collapsed about 70 per cent. Cavity closed and forms small oval homogeneously dense focus, 1.5 cm. x 1 cm. Heavy adhesion band from closed cavity to chest wall. Intrapleural pneumonolysis attempted 4/8/45 but adhesions could not be cut. Good deal of resolution in right lung lesion. Sputum negative.

There was much contralateral pathology in this case and the success in the closure of the tension cavity with a small pneumothorax collapse was very important. Otherwise the patient would have had a difficult therapeutic problem and a poor prognosis.

There were 4 other patients observed similar to Case 5. These individuals all had typical tension cavities prior to pneumothorax. The cavities were large, the diameters measuring 2½ cm., 3 cm., 4½ cm., and 5½ cm. respectively. They were spherical in shape and all but one had a thin wall. In 3 instances there was practically no infiltration surrounding the cavity and in one case the infiltration was very slight, although 2 of the patients had mixed infiltration in other areas of the homolateral lung. Contralateral productive infiltration was present in two of the patients. Cavity fluid levels were present in all before pneumothorax. In two of the patients a well-defined draining bronchus could be seen extending from the hilus area to the inferior and medial border of the cavity, with narrowing at the broncho-cavity junction demonstrated in one of them.

Two of the cavities were located in the right upper lobe; one was in the left upper lobe and one in the right lower lobe. The pneumothorax in each case was maintained with the use of subatmospheric pleural pressures.

Only a partial peripheral pneumothorax collapse was obtained for the lower lobe cavity. All of the upper lobes showed the apices adherent with many adhesions over the cavity area. Intrapleural pneumonolyses were attempted in all of the cases. In only one separation of adhesions was possible and this only slightly improved the pneumothorax space. The total collapse of the upper lobes was estimated to be about 20 per cent, 25 per cent, and 35 per cent, respectively. The point to be emphasized is that only a slight collapse of the lung was obtained for a very large cavity.

The complete closure of the cavities was definitely demonstrable within 5 weeks, 2½ months, 5 months and 7 months respectively. Before cavity closure, fluid levels were apparent in 3 of the 4 patients.

The sputum turned negative at intervals of 1 month, 7 weeks, 4 months and 4½ months after the initiation of pneumothorax. A negative sputum means that sputum concentrates, gastric concentrates and cultures were negative. These patients remained negative for 10, 11, 12, and 20 months' observation respectively.

The cavity at the time of closure appeared as a small, homogeneous, round focus twice and in the other two, only minimal nodular and linear infiltration remained. The closure of these cavities with pneumothorax avoided the need for more aggressive surgical procedures and preserved a maximum of good lung. This was doubly important as some patients could not have surgery done, and even with pneumothorax could tolerate only a slight collapse.

The roentgenograms showed definite evidence of valvular bronchial obstruction in these patients before and during pneumothorax therapy. There was rapid closure of these large tension cavities with a small pneumothorax collapse. This is explainable, as in Case 5, on the basis of closure of the draining bronchus which would remove the check-valve mechanism and cause collapse of the cavity.

It has been emphasized in several reports (Newton,[17] Hurst and Schwartz,[18] and Thompson and Greenberg[19]) that adhesions attached to diseased areas of the lung should be released, if possible, because of their responsibility for exacerbations and relapses, particularly after re-expansion. A perfect selective or anatomic collapse of the diseased part is the criterion and not alone conversion of the sputum and closure of cavities. In other words, to avoid poor, late results, an effective collapse must also be a complete one without adhesions.

We believe that adhesions should not be disregarded. We recommend the consideration of intrapleural pneumonolyses for adhesions over areas of pathology that prevent satisfactory collapse of diseased lung even in negative cases. In these patients of Group 4 intrapleural pneumonolyses were attempted but were unsuccessful. There was then the choice to re-expand the pneumothorax that though anatomically poor, had closed the cavity and converted the sputum, or to use another surgical procedure. Surgical therapy was, however, not always possible or was definitely contraindicated.

In these cases also there was no use of positive pressure pneumothorax with its deleterious effects. In tension cavities the actual amount of tissue destruction is much less than the size of the cavity indicates and therefore a partial pneumothorax may be adequate and control the disease. With these considerations in mind, the usual views about the hazards of incomplete collapse may be modified for these tension cavities.

Discussion

This paper is not a plea for the use of pneumothorax in the treatment of tension cavities. Its purpose is to describe the conditions under which pneumothorax can be successful in the closure of tension cavities. These cavities frequently cause failures in collapse therapy, particularly pneumothorax, and their control is of importance. This consideration is of special significance in those patients for whom thoracic surgery (the accepted form of treatment) is contraindicated and pneumothorax is desirable. Appreciating its limitations and infrequency, it is of value to know that pneumothorax can close tension cavities. We attempted in this study to determine the factors responsible in those cases where pneumothorax was effective. The follow-up period after closure of some of the cavities described has not been too long. Better evaluation of the treatment and the permanence of cavity closure will be possible after a longer observation period. This is important as cavities can re-open. Nevertheless, the successful methods

employed in these cases are worthy of trial in pneumothorax therapy.

The pneumothorax had to be modified in the following manner to obtain cavity closure: (1) A markedly collpased lung had to be considerably re-expanded and then refills were re-instituted with an increase of the collapse again obtained; (2) a markedly collapsed lung was considerably re-expanded and the pneumothorax was then maintained in this condition of partial collapse; (3) only a slight pneumothorax collapse was obtained after initiation and this was continued throughout the course of therapy.

Tension cavities are caused by valvular obstruction in the draining bronchus. As mentioned above, there are conditions in the lumen of the bronchus or its wall that can be responsible for the partial obstruction. In addition, the pneumothorax changes the direction and angulation of the bronchus, alters the size of the bronchial lumen and modifies the amount of cavity drainage. Variations in the degree of pneumothorax can therefore create, accentuate or remove a bronchial check-valve mechanism. In the cases described it was noticed that not every cavity had typical characteristics of tension prior to pneumothorax. However, after collapse was started the appearance of the marked tension cavities showed that the pneumothorax was responsible for the formation of the tension by bronchial changes. Cavity fluid levels were more often seen after pneumothorax was initiated, indicating that the pneumothorax made the bronchial drainage less efficient.

The effectiveness of pneumothorax depends upon many factors in the cavity, lung and pleural space, but especially the condition of the draining bronchus for if a valvular obstruction exists that creates a tension cavity, closure will not occur. The closure of the tension cavities obtained with the above modifications in the degree of pneumothorax is explained as being due to a release of the valvular bronchial obstruction with a resultant opening or closing of the draining bronchus and under these circumstances cavity collapse occurred.

Ordinarily one would expect re-expansion to cause opening of the bronchi. It may be considered paradoxical to state that bronchial closure will occur with lung expansion. As mentioned above, there are many conditions in the bronchial lumen or wall responsible for a valvular obstruction. It is possible for a partial obstruction because of the bronchial changes (angulation, drainage, etc.) that occur in expansion to close with a small degree of pneumothorax and to persist with a marked pneumothorax. We have shown that tension cavities closed with a small pneumothorax and in the Group 4 cases, evidence indicated that only the mechanism of bronchial closure was responsible.

The cases presented also demonstrated the importance of individualization in the pneumothorax therapy. Pneumothorax is a complex, skilled procedure and much more than merely introducing air into the pleural space to get a maximum amount of collapse. Each pneumothorax has its optimum collapse required for cavity closure. A selective collapse can be a partial or marked one. This satisfactory degree of collapse must be determined for each patient. The type of collapse (particularly in relation to changes in the draining bronchus) is the important consideration and not the amount of collapse per se. Although a selective collapse is not a matter of choice but depends upon the changes within the lung, still the degree of collapse can be regulated, and as illustrated by our cases, proper variations in collapse can determine whether the pneumothorax will be successful or not.

Many cavities gradually diminish in size as the pneumothorax is increased, and close only with a considerable collapse. Moreover, not all tension cavities will respond to re-expansion as did the cases described in this paper. It is probable, however, that some pneumothoraces are collapsed too fast and too much. By slowly increasing the pneumothorax and carefully observing changes in the cavity and sputum, a point may be reached where the therapy will be effective with less collapse than usually used. The most desirable degree of pneumothorax is the smallest amount which will close the cavity and control the diseased area and keep uncollapsed a maximum of good lung.

Re-expansion of the lung and modification of the degree of collapse, as suggested in this series, should be considered early. It is undesirable to continue an ineffective pneumothorax for a long time. Unfortunately, unsatisfactory pneumothoraces are too often maintained longer than they should. There should be no delay in any necessary surgical procedure because the closure of tension cavities with changes in the pneumothorax collapse becomes rapidly evident. It is desirable, prior to thoracoplasty, to have the lung completely re-expanded. Before the operation is done the sputum and roentgenogram should be re-checked because of the possibility of cavity closure with re-expansion. Likewise, whenever ineffective pneumothoraces are stopped, the lung should be carefully observed during re-expansion for changes in the cavity.

In some cases (as in Cases 1 and 2) with huge tension cavities, and the lung markedly collapsed, the expansion was much better than could be hoped for from examination of the roentgenogram. The actual destruction of lung was much less than indicated by size of the tension cavity. With removal of the tension and positive pressure from within the cavity the lung tissue around the cavity,

which had been compressed by this tension, expanded readily and filled in the space left by the closing cavity.

There was an interesting correlation between the sputum and x-ray findings. Frequently the sputum concentrates turned negative before the cavity was completely closed on roentgenogram. An ascending order of sensitivity in the examinations used for finding tubercle bacilli was also indicated, for in many of the cases with closure, sputum concentrates were converted first, the gastric concentrates next, and the cultures were last to turn negative.

CONCLUSION

Cases illustrating the closure of tension cavities with pneumothorax have been presented. In order to achieve these results the pneumothorax was modified by (a) considerable re-expansion, then increase of collapse; (b) considerable or partial re-expansion, and (c) a small degree of collapse. The relationship of the pneumothorax to the formation and closure of tension cavities has been discussed. The role of the draining bronchus in these processes has been described and emphasized.

CONCLUSION

Se han presentado casos ilustrativos del cierre de cavernas de tensión mediante el neumotórax. A fin de obtener estos resultados se modificó el neumotórax en las formas siguientes: (a) considerable reexpansión seguida de aumento del colapso; (b) considerable reexpansión y (c) pequeño grado de colapso. Se ha discutido la relación entre el neumotórax y la formación y el cierre de cavernas de tensión. Se ha descrito y recalcado el papel que desempeña en estos procesos el bronquio de desagüe.

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Discussion

BENJAMIN L. BROCK, M.D., F.C.C.P.
Oteen, North Carolina

I would like to discuss briefly the physiology of the bronchial tree, particularly as it relates to the pathogenesis of pulmonary tuberculosis. It has long been my belief that lack of adequate drainage from the lungs and bronchial tree plays a major role in the pathogenesis of this disease. Drainage of exudates from the bronchial tree is accomplished in the following ways:

(1) Through the ciliary action.
(2) Through the cough mechanism, and
(3) Through a peristalsis like action of the bronchial tree during respiration. During inspiration the bronchial tree elongates and the diameters of the lumina of the bronchi become wider. During expiration the bronchi shorten and the lumina are narrower.

This latter mechanism plays a large part in the evacuation of exudates which may accumulate within the bronchial tree and where adequate drainage is instituted disease may be prevented, or where disease already exists, it may clear.

To illustrate very clearly the part that drainage plays in the clearing of a tuberculous lesion we may consider the sequence of events which take place when endobronchial tuberculosis develops

to such an extent that partial blockage of the draining bronchus occurs. Under these conditions a marked retrograde spread of disease within the lung distal to the blockage takes place. This is purely mechanical and has nothing to do with lack of immunity of the individual.

Elevation of the foot of the bed will aid greatly in expelling exudate from the bronchial tree in upper lobe lesions. The rise of the diaphragm and its increased mobility equalize the movement of the lung in the otherwise less mobile apex and the peristalsis like action of the bronchial tree in this region initiates drainage of exudate which ordinarily would have remained intact.

The apex of the lung, particularly that portion situated in the costo-vertebral guttur, is the usual location where tuberculosis develops and progresses and where cavity formation frequently occurs.

It has long been stated that when an individual is put to bed because of active pulmonary tuberculosis his lesion clears because his lung is put at rest.

The metabolic rate of the individual is kept at a minimum under these conditions which is helpful in any type of infectious process, but his lungs are more active when he is lying down than when he is in a standing position. Drainage may be more adequate therefore when the patient is lying flat and still more adequate when the foot of his bed is elevated. This latter statement holds true where blockage of the bronchial tree takes place by exudate alone. Where anatomical blockage exists, as in endobronchial tuberculosis, these positions make the drainage more difficult and symptoms such as cough may increase. This may be explained by the fact that on expiration the bronchial tree becomes shorter and in addition the lumina of the bronchi become narrower. Under these conditions the blockage around the endobronchial lesion becomes more complete during expiration and the exudate distally will no longer pass through the bronchus.

Many of us have taken the attitude that artificial pneumothorax is contraindicated where obstructive endobronchial disease is present and particularly where tension cavities exist. It is true that many such cavities will close by allowing the lung to re-expand after pneumothorax has been established, but it is also true that many of these cavities will not close under these conditions. When the lung is collapsed in the presence of obstructive endobronchial disease, atelectasis is a common complication and it is usually impossible to get the lung re-expanded. Where tension cavity is present artificial pneumothorax simply makes matters worse. Compression over the cavity has a tendency to narrow the already partially blocked bronchus leading to the cavity, with the result

that the blockage becomes more complete, the tension cavity becomes larger and under greater tension. Air can still enter the cavity on inspiration, but on expiration the bronchiole through shortening and further narrowing will not allow air to escape. It is believed that in allowing the lung to re-expand the lumen of the bronchus entering the cavity becomes wider and because of the establishment of adequate drainage the cavity eventually closes.

In my opinion more cavities close because of good drainage than because of complete blockage with absorption of the air within the cavity.

The secret of any form of collapse therapy is the institution of adequate drainage. Artificial pneumothorax is successful because it collapses the lung and pushes out exudate which could not be evacuated otherwise. In other words, this establishes drainage and prevents retrograde spread of disease. It is not the splinting of the lung and putting it at rest which is important, as has so often been said.

Pneumoperitoneum has a remarkable effect on the closure of tension cavities. When a phrenemphaxis is done on one side, followed by pneumoperitoneum, two things are accomplished: First, the high rise of the diaphragm compresses the lung and thereby facilitates drainage. Second, there is a marked relaxation of the lung and bronchial tree. There is little shortening of the bronchi and little narrowing of their lumina during expiration and this allows for continuous drainage, through patent bronchi. This also allows for healing of the endobronchial lesion.

With the introduction of pneumoperitoneum in acute bilateral advanced disease, with tension cavity formation, in the negro, drainage may be made so adequate that the disease may clear entirely. In my opinion, such disease in the negro has been looked upon as fatal and such a result could not have been obtained by any other method of collapse therapy at our disposal.

Such results should modify our beliefs relative to the pathogenesis of pulmonary tuberculosis. Certainly a large factor in the spread of tuberculosis in the negro is his much greater allergic response to tubercule bacilli of reinfection with subsequent inability of the bronchial tree to institute adequate drainage of the exudate from the large immobile pneumonic areas.

Pneumoperitoneum with Diaphragmatic Paralysis in the Treatment of Pulmonary Tuberculosis

DAVID J. GILMORE, M.D.*
Detroit, Michigan

Pneumoperitoneum has been used in the treatment of pulmonary tuberculosis since 1931.[1] However, there still exist sharp differences of opinion as to its efficacy and safety. Because our results in difficult cases have been gratifying and because certain hitherto unpublished observations have been made, we feel that our experiences should prove interesting, although the number of cases treated is small.

Method

In each of the cases treated with pneumoperitoneum in the Henry Ford Hospital, the diaphragm was examined fluoroscopically to avoid the selection of patients whose diaphragms were fixed by adhesions. Then, prior to the injection of air, the phrenic nerve was crushed on the side to be treated, and the diaphragmatic paralysis confirmed by fluoroscopic examination. Air was introduced in the manner described in detail by several authors.[2,3] The technique was essentially that used in instituting and refilling pneumothoraces; the pneumothorax apparatus was used. The site of introduction was usually two to three inches to the left and about an inch below the level of the umbilicus. (Occasionally a point in the left upper quadrant was chosen). From 300 to 600 cc. of air were introduced in the first injection. Three to four "refills" were given at two or three day intervals, increasing each injection by 100 or 200 cc. The rapidity with which the amounts of air were increased depended upon the patient's tolerance and the evidences of diaphragmatic elevation observed fluoroscopically and by means of chest roentgenograms. The shoulder and epigastric distress following the first few injections was usually transient, and never severe. After a few injections the intervals were prolonged and eventually averaged 12 days (varying from 10 to 20 days). The average refill was 1100 cc. (varying from 600 cc. to 1350 cc.), and the average intraperitoneal pressure was +10 cm. of water. (The pressure varied from +8 to +14 cm. of water).

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*From the Cardio-Respiratory Division, Department of Medicine, Henry Ford Hospital, Detroit 2, Michigan.

Indications

The indications for artificial pneumoperitoneum in pulmonary tuberculosis have been discussed at length by other observers.[4-7] We have used it *only* following phrenic interruption by crushing. (However, in one case pneumoperitoneum was continued in spite of an ineffective phrenic crushing (Table 1, No. 3). In *all* of the cases reported in this group, pneumothoraces were attempted but abandoned because of extensive adhesions; pneumonolyses were considered impractical and unsafe. Since the group includes only cases with far advanced and moderately advanced pulmonary tuberculosis, a major indication was preparation for ultimate thoracoplasty. As will be further noted, the unexpected and marked improvement in several made the radical surgery unnecessary. In one case, pneumoperitoneum was instituted because the patient refused to submit to thoracoplasty. In another, massive hemoptysis caused us to use the measure as an "emergency treatment"; in this case, of course, there were other indications for the procedure. Because of the severity of the disease in all of the twelve cases, longer periods of observation without some form of collapse therapy were considered unsafe.

Diaphragmatic Elevation

Emphasis has been placed on the greater elevation of the hemi-diaphragm obtainable with pneumoperitoneum following phrenic nerve interruption than with phrenic paralysis alone.[8,9] In the present series, the average elevation of the paralyzed hemi-diaphragm

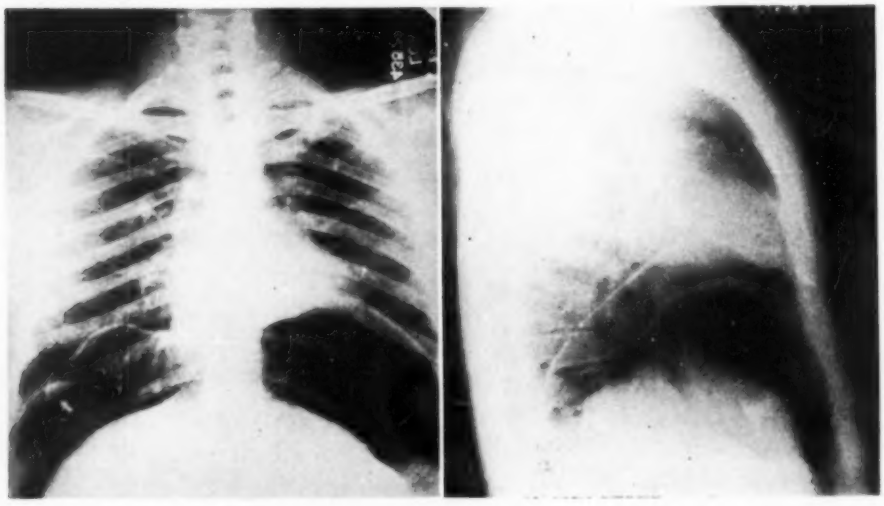

Figure 1 Figure 2

Fig. 1 and 2 (Case No. 8): Note the configuration of the elevated diaphragm and compare with Figure 5.

following phrenic crushings was 2 cm.; the average elevation following pneumoperitoneum in the same patients was 8 cm.; this represents an average added elevation of 6 cm. The unparalyzed hemi-diaphragms usually did not rise, although in several cases elevations of 2 or 3 cm. resulted. The measurements were made on roentgenograms taken with the patients upright in the postero-anterior position. We wish to point out that the diaphragmatic elevations determined in this manner may give entirely false impressions as to the magnitude of the decrease in lung volume. The paralyzed diaphragm rising as the result of alterations in the relationships between intrathoracic and intrabdominal pressures frequently does not do so as a unit. Hence, though the postero-anterior view may display what seems to be marked and diffuse elevation, a lateral view may reveal that the elevation is confined to one portion of the hemi-diaphragm. Figures 4 and 5 illustrate what may be considered ideal diaphragmatic elevation. In Figures 1 and 2 the point just stated is illustrated. In the postero-anterior view, the left hemi-diaphragm seems to be fairly well elevated. In the lateral view (Fig. 2) it is seen that the diaphragmatic rise is confined to the anterior portion. In some cases, the lack of uniformity is more marked than this. Therefore, it is suggested that the volume of collapse be determined by examining the hemi-diaphragms in the *two* positions. Such observations may help in making the often difficult decision as to whether the pneumoperitoneum should be abandoned or continued in those cases in which conclusive improvement does not occur in a reasonable period.

Results

Twelve cases treated with pneumoperitoneum supplementing phrenic paralysis are summarized in the accompanying table. Six of these had far advanced pulmonary tuberculosis; six moderately advanced. The two (Nos. 5 and 6) classified as "poor" results were the only ones in which the therapy was considered to have failed. The results in three (Nos. 3, 9 and 12) were considered "fair," indicating that, although there was definite improvement, either complications prohibited continuing or all the criteria of improvement were not met. The remaining seven were classified as "excellent" results. In these marked improvement resulted as measured by the clinical course (disappearance of symptoms and fever with a steady weight gain), serial roentgenograms, sedimentation rate decreases, and the disappearance of tubercle bacilli from the sputum.

Certain significant facts merit emphasis. Nine cases were treated with pneumoperitoneum and phrenic paralysis *only* to prepare

for radical surgical measures. Eight of these improved so markedly and rapidly that thoracoplasties were eventually unnecessary. In five of the group of twelve sputum specimens became "negative" in three months or less. Cavities in the *upper thirds* of the lung fields were closed in seven cases out of ten. The excellent results obtained in Case No. 10 are illustrated by x-ray films (Figs. 6 and 7), which show the closure of two large cavities. X-ray films taken seven months apart (Figs. 3 and 4), show the closure of an apical cavity in Case No. 11. Attention is also directed to the fact that

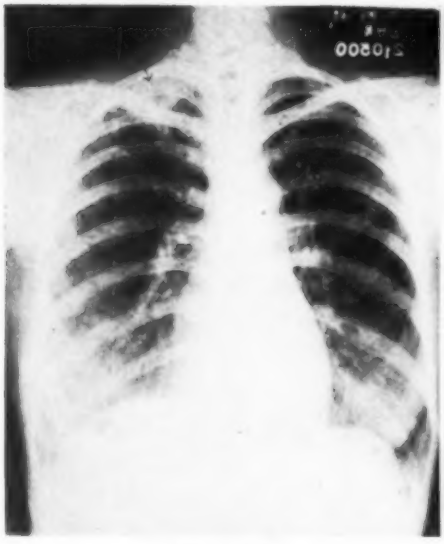

Figure 3

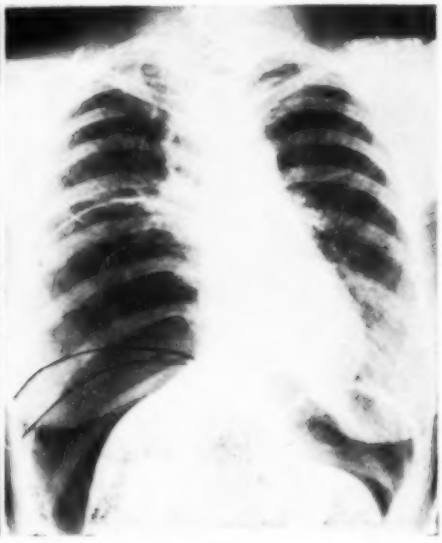

Figure 4

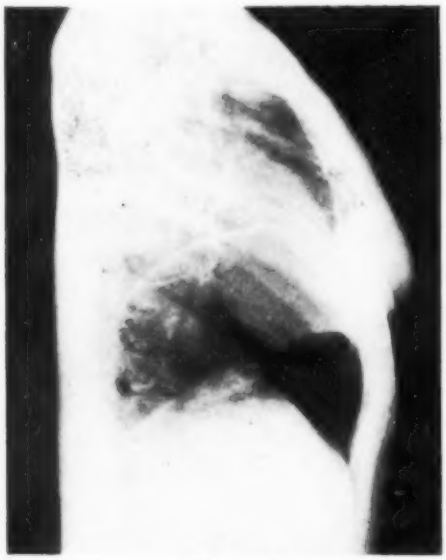

Figure 5

Fig. 3 and 4 (Case No. 11): The lower black line represents the level of the diaphragm prior to any therapy; the upper black line, the level after phrenic paralysis.

Fig. 5 (Case No. 11): Compare the diaphragmatic configuration with that in Fig. 2. (See text for discussion).

effective diaphragmatic elevations have been maintained in three ambulatory patients (Nos. 1, 2 and 7, for 18, 8 and 9 months, respectively).

Complications

In addition to transient and mild epigastric and shoulder pain, other complications encountered deserve attention. As is noted in Table 1, patient No. 4 developed a small umbilical hernia. However, this caused no symptoms and did not interfere with the continuation of the therapy. In two cases (Nos. 6 and 9), anorexia and nausea with vomiting forced us to abandon the pneumoperitoneums. It is noteworthy that the symptoms appeared only after months had elapsed. It has been our feeling that strong emotional factors were contributory in these cases. The 12th case (Table 1), represents a complication of the axillary route of air injection. In this case, diaphragmatic elevation seemed to be sufficiently high to permit the use of a site in the lower axilla. The result, as described, was an accidental pneumothorax. Though the pleural adhesions had led us to believe pneumonolysis an unsafe procedure in the beginning, the accidental introduction of a large amount of air into the pleural space revealed a less formidable picture. It was then decided to cut the adhesions and attempt to continue the pneumothorax. The results indicated that our original opinion relative to pneumonolysis was correct. No more serious complications were encountered in the eighteen cases in

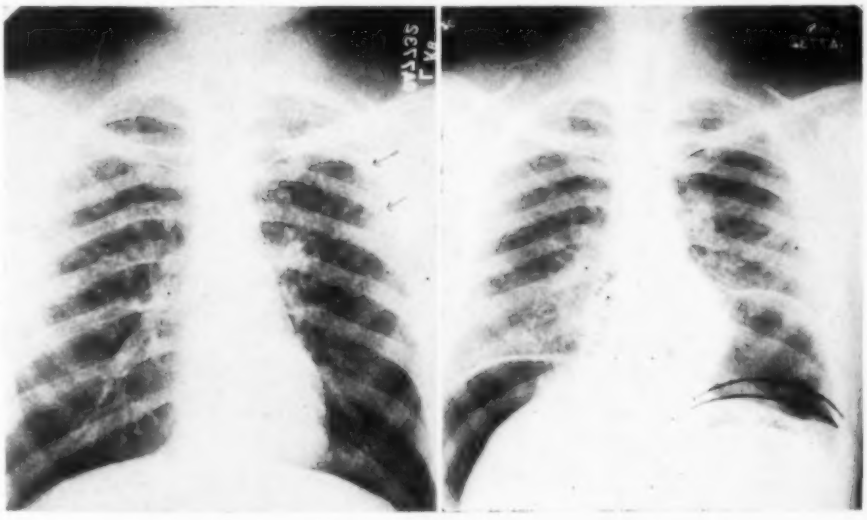

Figure 6 Figure 7

Fig. 6 and 7 (Case No. 10): The black lines have the same significance as in Fig. 2 and Fig. 4. Note the disappearance of the two cavities.

TABLE ONE

No., Age, Lung	*Severity of pulmonary disease*	*Course prior to Pneumoperitoneum (PNP)*	*Interval between phrenic and PNP*	*Duration of PNP*	*Results; comments regarding course and complications*
(1) 20 yrs. RIGHT	III B Bilateral; cavity in right apex, 3 cm. in diameter.	Symptoms 5 yrs.; pneumothorax tried on right; no space. Thoracoplasty deferred. On bed rest alone: temp. dropped to normal and wt. up 25 lbs. Sputum POSITIVE (in spite of rt. phrenic); Sed. rate 6 mm/hr.	14 weeks Right phrenic nerve crushed.	4 years	Results: EXCELLENT. Undertaken to prepare for thoracoplasty; sputum NEGATIVE in 1 month; cavity closed in few months. Ambulatory 18 months; axillary refills. Cavity appeared in *left* upper four years after PNP. started; PNP. therefore discontinued.
(2) 20 yrs. LEFT	III B Bilateral; cavity middle third, left, c cm.	Prognosis poor; severe tuberculous enteritis; thoracoplasty deferred; left pneumothorax attempted, adhesions prohibited continuing. For 3 mos. wt. loss, diarrhea, x-ray films same. Sputum POSITIVE; sed. rate 44.	3 weeks Left phrenic nerve crushed.	30 months continues.	Result: EXCELLENT. Gastrointestinal symptoms relieved in 3 mos.; gained 14 lbs. in 3 mos.; sputum NEGATIVE in 1 yr.; after 7 mos. thoracoplasty advised but refused; progress excellent. Ambulatory 8 months.
(3) 20 yrs. RIGHT	III B Bilateral; upper lung fields.	Prognosis poor; right pneumothorax complicated by tuberculous empyema; at time of PNP.: reactivation both sides. Sed. rate 42; sputum NEGATIVE.	Right crushed twice; third attempt failed.	19 months continues.	Result: FAIR. Delayed but definite improvement in lesion on right though at first more extensive; persistently high sed. rate; no weight gain. In spite pneumothorax, left not improving satisfactorily.
(4) 39 yrs. RIGHT	III B Bilateral; cavities both apices Rt. 3 cm. diameter.	Prognosis poor. Sputum POSITIVE. Sed. rate 38 mm/hr. Wt. 84 lbs. marked symptoms. Right pneumothorax attempted but pleura adherent.	3 days Right nerve crushed.	15 months continues.	Result: EXCELLENT. Sputum NEGATIVE in 2 mos.; wt. gain 20 lbs. X-ray: cavity apparently closed in 4 mos.; in 1 yr., right lesion very small, fibrotic; left improved. Small umbilical hernia; multiparous.
(5) 19 yrs. RIGHT	III B Cavity rt. 2nd rib 2 cm. in diameter.	Prognosis poor. Treatment 7 yrs.; bilateral pneumothoraces; rt. until 3 yrs. ago; left to date of PNP. Pneumothorax attempted again: no space. Thoracoplasty advised; refused. Sputum NEGATIVE; sed. rate 46.	7 weeks Right nerve crushed.	14 months.	Result: POOR. Cavity "apparently closed" and then "reopened" in the course of a year. PNP. abandoned; thoracoplasty if patient agrees. Sputum NEGATIVE; sed. rate 39 mm/hr.
(6)	III B Bilateral;	Right pneumothorax attempted and abandoned after a few days — no	6 days Right	10 months.	Result: POOR. Cavity appeared left side in 1 month.

(6) 22 yrs. RIGHT	III B Bilateral; cavity rt. apex 2 cm. in diam.	Right pneumothorax attempted and abandoned after a few days — no pleural space. Sputum POSITIVE. Sed rate 50 mm/hr. Symptoms severe at first.	6 days Right nerve crushed.	10 months.	Result: POOR. Cavity appeared left side in 1 month. Nausea and vomiting forced discontinuation PNP.; sputum still POSITIVE; sed. rate 44 mm/hr. X-ray not improved.
(7) 28 yrs. LEFT	II B Cavity 2nd rib left 2.5 cm. diameter.	Pneumothorax not feasible; extensive pleural adhesions. On bed rest and with phrenic, wt. gain and sputum NEGATIVE, but no x-ray change in 5 mos. Thoracoplasty deemed "inevitable". Sed. rate 34 mm/hr. Wt. 135 lbs.	4 months Left phrenic crushed. Repeated 6 days before PNP	24 months continues.	Result: EXCELLENT. X-ray revealed definite improvement in 4 months; in 6 months cavity not seen. Wt. 175 lbs. Sed. rate 11 mm/hr. Abdominal binder used at first. Ambulatory 9 mos.; back at work.
(8) 29 yrs. LEFT	II B Cavity 1st rib left 1.5 cm. diameter.	Left pneumothorax attempted; pneumonolysis failed; abandoned. Two months after phrenic still had POSITIVE sputum; slight wt. gain. slight improvement in x-ray. Sed. rate 30 mm/hr.	2 months Left nerve crushed.	10 months continues.	Result: EXCELLENT. Sputum NEGATIVE 1 mo.; wt. gain 30 lbs.; sed. rate 15 mm/hr. X-ray: definite clearing in two mos.; cavity "closed" since 4 mos. following institution of PNP.
(9) 21 yrs. RIGHT	II B Cavity at level right 2nd rib 2 cm. in diameter.	Previous pneumothorax for left cavity; left inactive recently. Rt. pnthx. attempted: no space. Massive hemoptysis few hours before PNP. started. Sputum POSITIVE; sed. rate 5 mm/hr.	2 weeks Right phrenic nerve crushed.	7 months.	Result: FAIR. Hemoptysis controlled; in 3 mos. sputum NEGATIVE; 2 mos.: cavity not seen; wt. gain 5 mos., then anorexia forced abandon PNP. Thoracoplasty refused; improved; discharged; fair progress.
(10) 28 yrs. LEFT	II B 2 cavities 2nd rib 3.5 and 2.5 cm. in diameter.	Thoracoplasty deemed "inevitable". Pneumothorax tried: no space. Following phrenic, gain in wt. but x-ray film same; sed. rate still 30 mm., and gastric washings POSITIVE.	5 weeks Left phrenic nerve crushed.	10 months continues.	Result: EXCELLENT. Continued wt. gain; sed. rate to 5 mm/hr. X-ray: cavities not distinguished after 6 mos.; "closed" at 8 mos.
(11) 45 yrs. RIGHT	II B Cavity 1.5 cm. in diameter, rt. apex.	Pneumothorax attempted on right; extensive adhesions prevented. Sputum NEGATIVE; sed. rate 43. Wt. loss continued in presence phrenic and strict bed rest. Thoracoplasty deemed "inevitable".	1 month Right phrenic nerve crushed.	7 months continues.	Result: EXCELLENT. MARKED x-ray improvement; cavity "closed" after 5 mos.; sed. rate to 23 mm/hr. in 2 mos.; wt gain of 7 lbs.
(12) 24 yrs. RIGHT	II B Cavities above 2nd rt. rib—3 cm., 1 cm. and 1 cm. in diam.	Right pneumothorax tried; adhesions prevented safe continuation; pneumonolysis considered unsafe. Sputum POSITIVE; sed. rate 30 mm/hr. X-ray not improved after two mos. of bed rest with phrenic. Thoracoplasty planned as ultimate therapy.	6 weeks Right phrenic nerve crushed.	5 months.	Result: FAIR—though period too short for proper evaluation, during PNP.; sputum NEGATIVE (3 mos.); wt. gain; x-ray improved. Accidental pneumothorax on right; pneumonolysis; empyema developed; rib resection; refused further surgery; progress now fair; sputum still negative; PNP. not restarted.

which pneumoperitoneum has been used. (The six cases not reported at this time were omitted because the periods of therapy were less than four months).

Comments

It was noted above that, in spite of the maximum doses of air given, the unparalyzed hemidiaphragms were rarely elevated and that the elevation that did occur was slight. It is apparent that the volume of collapse cannot be significant if the diaphragm does not rise. It is our belief that, to obtain the greatest benefits, pneumoperitoneum must be used *following* phrenic interruption. The therapeutic value of the measure should not be appraised by reviewing results obtained in the presence of unparalyzed hemidiaphragms.

The statistics of the small number of cases in this report may not seem significant. However, as is suggested by the case summaries presented in the accompanying table, the courses followed by these patients have been most impressive. In each of nine we were forced to plan thoracoplasties as the ultimate therapeutic step. We watched eight of these improve with relative rapidity and finally arrive at stages of healing and arrest which made surgical "loss" of lung tissue no longer appear necessary. Such an experience causes us to urge the careful consideration of the use of this method of therapy in selected cases before drastic measures are instituted.

SUMMARY AND CONCLUSIONS

The histories and the results and complications of therapy in twelve cases of pulmonary tuberculosis treated with pneumoperitoneum and phrenic paralysis were tabulated and discussed:

1. Of these, six were far advanced cases of pulmonary tuberculosis and six moderately advanced.

2. The results were *excellent* in 7 (58 per cent); *fair* in 3 (25 per cent) and *poor* in 2 (17 per cent).

3. Cavities in the upper thirds of the lung fields were closed in 9 out of 11 cases.

4. In 8 out of 9 cases thoracoplasties were avoided by the use of pneumoperitoneum with hemi-diaphragmatic paralysis.

5. It was suggested that the estimation of diaphragmatic elevation and the volume of lung collapsed should be based on lateral as well as postero-anterior roentgenologic examination of the chest.

6. Pneumoperitoneum alone does not result in effective diaphragmatic elevation; it should be used with phrenic interruption to obtain the greatest benefits.

RESUMEN Y CONCLUSIONES

Se presentan en forma tabular y se discuten las historias, los resultados y las complicaciones de la terapia en doce casos de tuberculosis pulmonar tratados mediante el neumoperitoneo y la parálisis del frénico.

1. De estos, seis fueron casos muy avanzados de tuberculosis pulmonar y seis moderadamente avanzados.

2. Los resultados fueron *excelentes* en 7 (58 por ciento); *regulares* en 3 (25 por ciento) y *malos* en 2 (17 por ciento).

3. De 11 casos, en 9 se cerraron cavernas en los tercios superiores de los campos pulmonares.

4. De 9 casos, en 8 se evitaron toracoplastias mediante el uso del neumoperitoneo con la parálisis hemidiafragmática.

5. Se sugiere que el cálculo de la elevación diafragmática y del volumen de pulmón colapsado debe estar basado en exámenes roentgenológicos del tórax, tanto laterales como postero-anteriores.

6. El neumoperitoneo por sí solo no causa elevación eficaz del diafragma; debe ser empleado en combinación con la interrupción del frénico para obtener mayores beneficios.

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EDITORIAL

THE THIRTEENTH ANNUAL MEETING

Another milestone will be dedicated along the route of progress of the American College of Chest Physicians with the opening of the thirteenth annual convention of this organization. On this occasion, it is fascinating to recall how the College was borne from an idea of a few enthusiastic chest specialists and how it has reached from its inauspicious beginnings the stage where its function in the realm of medical science, education and in other fields of specialized activities is of importance to the profession and to the public throughout the civilized world. In retrospect, it is with great satisfaction and with excusable pride for each and every one of the members of the College to see that their individual efforts, loyalty and solidarity are bearing precious fruits in the form of widely appreciated achievements of this Society.

The scientific program of the next annual convention of the College, inspired by the youthful, dynamic and energetic ambition of its membership, is a symbolic expression of their collective thinking and endeavor. It is a notable embodiment of our ardent and sincere desire to serve, through science, our fellow practitioners of the healing arts and the community in need of competent, modern medical care.

Glancing over the Preliminary Program, it is at once obvious that it encompasses a great variety of subjects which come under the heading of chest diseases. Regardless of the particular preference of practice of the individual physician, he will find a great deal of material dealing directly with his own work or with subjects closely related to it.

The scientific transactions begin with papers devoted to the problem of tuberculosis. No apologies need be offered for this arrangement. Complacency and nonchalance are out of place in our attitude concerning this disease, in spite of the marvelous accomplishments in its diagnosis and treatment. Let us not forget that even today, one person is dying of tuberculosis in this country every ten minutes.

The relentless campaign for the early detection of tuberculosis is producing concrete results. A much higher number of cases with minimal disease are diagnosed today than ever before. This fact may soon bring about the necessity of a critical review of the therapeutic approach in these instances. It is known that a certain percentage of patients with minimal tuberculosis recover without

therapeutic intervention. This observation, however, must not for a moment be interpreted as advocating the abandonment of proper care in every case of active tuberculosis. As long as we have no reliable gauge at our disposal for ascertaining which of the early cases of tuberculosis will recover spontaneously and which will not, no arbitrary selection is permissible in this respect. We are obliged to follow the usual standard lines of treatment. But, are there uniformly accepted measures for the care of minimal cases?

Bed-rest has been given much emphasis during the past decade as a sine-qua-non measure. This concept developed partly as a more or less natural reaction to the "rustic" type of cure-taking practiced in the early days of sanatorium regimen. Partly, it owes its popularity to a clearer insight into the physiology and pathologic physiology of the lung. Even so, there are logical and competent objections to enforcing absolute bed-rest in all forms of pulmonary tuberculosis. Well-documented clinical observations offer a firm foundation for a sound scepticism in this regard. For this reason, we welcome the opportunity of listening to the discussion of "The Use and Abuse of Bed-rest in the Treatment of Tuberculosis."

There has been a great deal of controversy concerning the fundamental factors responsible for the healing process in tuberculosis of the lung. The pros and cons of the question of blood supply in the artificially relaxed lung are one of the items which in spite of tremendous amount of work spent on this problem, require further elucidation. In one of the presentations at the College meeting, illustrative lantern slides and motion pictures will be shown for the concrete demonstration of changes observed in the experimentally relaxed lung. Thus, through indefatigable research of many years, we will be in a position to settle this issue once and for all and remove it from the realm of confused speculation.

Two of the papers to be presented deal with certain difficulties which may arise in connection with the use of artificial pneumothorax. The latter is still the most widely used form of relaxation therapy. Therefore, whatever constructive information is made available in these presentations, it will serve as a welcome guide for all of us.

Another topic dealing with tuberculosis is given in the paper on "Extrapleural Pneumothorax." This procedure is looked upon as a useful measure for the treatment of tuberculosis in instances where the stage of the tuberculous process or other circumstances call for mechanical relaxation therapy but other methods are impossible, less applicable or actually contraindicated.

An analysis of the physiologic principles of artificial pneumo-

peritoneum has also been included in the program. Pneumoperitoneum treatment, originated in this country in 1931, is gradually gaining recognition. Nevertheless, in many medical minds, there is a lack of clear visualization of the train of events connected with its clinical application. No doubt, some of the hesitancy and disinclination to resort to its use in selected cases may be attributable to the absence of adequate information on this subject in available texts on tuberculosis. The dissertation scheduled on the basic mechanical factors of artificial pneumoperitoneum may bring about a lucid understanding and a better appreciation of the potential value of this treatment. A substantial number of patients treated with artificial pneumoperitoneum will be reviewed in another presentation. We may anticipate the clarification of significant pertinent items, such as the selection of cases, technical refinements, immediate clinical response, roentgenologic changes, late therapeutic results and other data. Undoubtedly, the observations to be presented will serve as a firm foundation for an unbiased evaluation of this procedure.

We are exceptionally fortunate in having the privilege of listening to a discussion of "Combined Thoracoplasty." Being presented by a Continental clinician, it brings together a critical review and innovations of the modern on the one hand and the reminiscences of the pioneering days of chest surgery, carried out by De Cerenville in Switzerland more than sixty years ago, on the other.

All those who have followed the expansion of thoracic surgery in the treatment of tuberculosis have encountered instances where in spite of the critical selection of cases and despite superior technique, thoracoplasty failed to establish an arrest of the disease. This particular shortcoming of thoracoplasty, together with auxiliary methods of treatment are going to be dealt with in a paper entitled "Surgical Treatment of Residual Cavities Following Thoracoplasty."

To Tuffier belongs the credit for being the first to perform pulmonary resection in 1891. Modern surgical technique and advanced clinical knowledge brought this method into the limelight during the past few years. Definite ideas have crystalized as to the indications for this form of surgery in pulmonary tuberculosis. Even so, the final place of this operation may not be established for some time. But the discussion of this subject at the convention by thoracic surgeons of wide experience will certainly contribute to the clarification of the issue. Also, some commentary on this problem is forthcoming in a general review to be given by another chest surgeon in a paper, "The Extremely Conservative and Radical Treatment of Tuberculosis."

No purview of the treatment of tuberculosis would be complete

without due consideration of rehabilitation. Whatever therapeutic approach is chosen, its ultimate aim is the rehabilitation of the patient not only physically but also socially, economically and psychologically. Let us not forget that the treatment, medical or surgical, is not concerned with the lungs alone but it must be concerned with a human being as a whole whose traits, desires, ambitions and aspirations are the same as our very own. He who fails to appreciate this, no matter how skillful a technician or wizard of a theoretician he might be, fails to treat his patient adequately.

The extraordinary accomplishment of the veterinary profession in the virtual eradication of tuberculosis in the cattle has focused the attention of all concerned on the possibility of a similar goal in the human. Their achievement prompts the question of initiating a vigorous campaign in our own midst. Of various laudable projects, it seems that the attack on tuberculosis in rural communities is attaining a hundred per cent victory. In this instance, the attack has been directed against a point of resistance where the incidence of tuberculosis morbidity and mortality were known to be the lowest. It is reasonable to hope that by breaking its weakest link, we will loosen the grip of the chain of tuberculosis as an endemic disease.

In no country in the world has there been a well-organized and well-conceived antituberculosis movement carried out by public health authorities on such a large scale as in the United States. Its planning had to be done on a gigantic scale. All branches of medical science, from bacteriology to roentgenology were sifted for the most reliable methods which could serve the purpose. Through its efficient, coordinated and allocated directives, the Tuberculosis Control Division of the United States Public Health Service already has evolved a system which is likely to solve this huge problem. It is gratifying to note that the United States Public Health Service considered it expedient to contribute to the transactions of the annual meeting of the College with the appearance on the program of two of its most outstanding experts in this field. It will be interesting to compare at the meeting data collected in this country with those recorded by a similar group of investigators in Paraguay.

The various facets of the complexities of the clinical and administrative management of tuberculosis will be reviewed by the English author of the historical study, "The Brompton Hospital; A Centenary Review." This panoramic view of changes concerning tuberculosis is bound to offer not only a magnificent historical perspective but also plenty of food for thought and endless incentive for further progress in our fight against this disease.

In harmony with the general scientific interest of the College, arrangements have been made for the presentation of a paper on Streptomycin Therapy in Tuberculosis. The exposition of this subject will be given by a representative of the United States Veterans Administration who is a member of the Streptomycin Committee of this organization. The latter with its excellent facilities and with the advantages of cooperative research is looked upon as a most potent source of modern scientific information. This circumstance endows this presentation with the hallamrk of highest competency.

As part of the scientific program, the originator of pulmonary decortication will present his experience with this procedure. We may look forward to a most enlightening and instructive exposition of this subject. No doubt, his illustrative cases will do away with some of the antagonism and scepticism in this regard and bring about a deservedly wider application of this method of treatment.

The achievements of thoracic surgery in the treatment of patent ductus arteriosus are meeting with uniform enthusiasm and admiration for the ingenuity and skill of the qualified chest surgeon. The painstaking experimental and clinical researches of the Johns Hopkins group are looked upon as cornerstones of more extensive surgical interventions in cardiovascular diseases. Sporadic case reports are gradually accumulating from all over the country as time goes on. A review of the present status of this operation, together with the personal experience of one of the speakers is scheduled on the program.

Speaking of cardiovascular diseases, reference may be made to another topic on the scientific program, namely to Ayerza's Disease. The author of this paper, the writer has been reliably informed, has seen more cases of Ayerza's disease than any other clinician in the world. His extensive observations and authoritative conclusions should be of value to all chest physicians.

Cancer of the lung could not have been omitted from this program. The menacing actuality of this disease becomes more and more obvious every day. The tremendous increase in the incidence of pulmonary cancer is real and not a matter of juggling with statistical figures. The annual death rate from carcinoma in general in the United States is 160,000. Considering that 10 per cent of all cancers are primary bronchogenic carcinomas, we must note that 16,000 people die of lung cancer in this country each year. The most tragic feature of this situation is the fact that at the time when the diagnosis of cancer is established, the condition is inoperable in more than one-half of the cases. At the convention, this pressing problem should be given all the attention its magnitude and seriousness demand.

Bronchiectasis will be presented at the meeting from the medical as well as from the surgical viewpoints. Bronchiectasis is a more common disease than it is realized by the medical profession. All cases of bronchiectasis, in the reversible phase of the disease are medical problems. When no adequate treatment is available at this stage, the chances for substantial improvement by medical measures are bound to decrease in proportion to the duration and severity of the lesion. It is the consensus that about 50 per cent of the chronic cases are eligible for surgical intervention. Up-to-date, first-hand information concerning the merit and value of surgery for bronchiectasis will be available for those attending the scientific sessions.

Each scientific presentation is to be followed by discussants assigned to this task and from the floor. These comments are bound to throw additional light upon the various subjects.

A brilliant array of speakers and worthy topics have been scheduled for the scientific program of the International Night. The participants include delegates from the Chapters of the College in Mexico, Puerto Rico, Cuba, Argentina and Paraguay. Tentative arrangements have been made for the participation in the scientific transactions of Fellows from China, the Philippine Islands and Australia.

Space does not permit fitting commentary on all of the subjects of the program. Suffice it to say that no effort has been spared to offer a well balanced program. Ample opportunity has been given to the discussion of diagnostic questions, such as "The Clinical Significance of Pulmonary Hemorrhage," "The Role of Bronchoscopy in Clinical Medicine and Surgery," "The Importance and Value of Bacteriologic Studies of Surgical Pathologic Specimens," "The Role of the Gastrointestinal Tract in the Production of Thoracic Symptoms," "Bronchography by Vaporization" and "The Clinical Application of Angiocardiography." The medical and surgical aspects of diseases of the lung and other thoracic organs are well covered and include such items as "Surgery of the Esophagus," "The Surgical Lesions of Coccidioidomycosis" and the "Significance of Solitary Mass in the Lung." Due time has been reserved for the presentation of nontuberculous diseases, such as "Meig's Syndrome," "Pulmonary Embolism as a Cause of Acute Coronary Insufficiency," "Asbestosis," "Bagassosis," "Cystic Disease of the Lung," 'Pancreatic Insufficiency in Early Life, with Special Reference to its Pulmonary Manifestations" and "Parasitic Infestations of the Lung." In addition, general reviews and progress notes will be offered by good speakers on "Pitfalls in Dealing with Cancer Statistics, Especially as Related to Cancer of the

Lung" and on "Recent Advances in Vitamin Research as Related to Clinical Medicine."

One of the outstanding features of the annual convention is an evening session set aside for "Information Please in Diseases of the Chest." The panel of Experts is composed of nationally recognized authorities and top-ranking scientists in their respective specialties, thus guaranteeing a most edifying and profitable scientific meeting.

Similarly, the annual X-Ray Conference will serve as a rich source of information concerning a variety of interesting problem cases.

Members of the College will get together at this convention from all parts of the United States and from abroad. They gather in an atmosphere of good-fellowship, mutual cooperation and the service of science. For the men with an open mind and of a receptive disposition, the annual meeting offers a gold mine of hitherto unpublished scientific information either through formal presentations or through informal personal contacts. We feel that seasoning one's thought's with the views of others is one of the best catalyzers for constructive, creative thinking.

It is hoped that the interest of the membership of the College in attending this meeting will be as great as the efforts given to its arrangement.

A. L. B.

REPORT ON STREPTOMYCIN IN TUBERCULOSIS

BY THE COMMITTEE ON CHEMOTHERAPY

Since the discovery of streptomycin by Waksman and his co-workers three years ago there has developed a tremendous interest in the therapeutic possibilities of this drug in the treatment of human tuberculosis. Feldman and Hinshaw have demonstrated conclusively that streptomycin has the remarkable ability to inhibit the development of tuberculosis in experimental animals. Their work has been confirmed by a number of other investigators.

The clinical investigation of streptomycin in the treatment of tuberculosis in humans was begun in the fall of 1944. Careful pharmacological studies were done to determine what deleterious effects or undesirable side reactions could be expected from its use. In the beginning cases selected for study were those in which the disease was progressing and routine sanatorium care had proved ineffective. With few exceptions patients were not included for study with the drug in which the prognosis was good or in which collapse therapy was feasible.

Progress of the investigation was necessarily slow at first due to limited supplies of the drug. As more and more patients were treated, it became apparent however that streptomycin did exert a suppressive effect on the progress of tuberculous infection in man as it had in experimental animals. It has a low toxicity for man and seldom did its administration have to be discontinued because of undesirable side reactions. There are at present a number of carefully conducted studies being carried out in different parts of the country to determine the effectiveness of streptomycin in the various types of human tuberculosis. The Committee on Chemotherapy has made an attempt to keep in touch with the progress of these studies.

With increased production the price of streptomycin has dropped steadily during the past year. At this time, January 1947, the quoted price is $4.80 per gram, making it available to more and more people. In view of this fact we felt it advisable to summarize briefly our impressions of the present status of the drug for the benefit of those physicians who may have occasion to use it.

It, of course, is not the purpose of this report to review in detail the clinical work which has been done with streptomycin. In recent months several papers have appeared in the medical literature reporting the clinical results of its use in human tuberculosis. From these published reports and from correspondence with other clinical investigators using streptomycin, some rather definite impressions have been obtained.

Streptomycin is undoubtedly a valuable adjunct in the treatment of human tuberculosis. At this time it cannot be recommended as a substitute for sanatorium care or other accepted therapeutic procedures in pulmonary tuberculosis. Results to date would seem to indicate, however, that its use may modify our therapeutic approach to certain types of cases. Advanced cases requiring thorocoplasty, for example, may perhaps be brought to surgery much sooner with streptomycin than with bed rest alone. Some cases may become suitable subjects for thorocoplasty who otherwise might never reach such status. Some chest surgeons are using streptomycin before and after resection for pulmonary tuberculosis in the hope that its use may prevent postoperative spread of the disease.

This is indeed a very fertile field for further investigation. It appears likely that a number of patients whose prognosis is considered good with routine bed rest may have their sanatorium stay considerably shortened by the use of this drug.

The status of streptomycin in the treatment of renal tuberculosis is equivocal at this time.

A few cases of proven tuberculous meningitis and miliary tuberculosis are still alive and apparently well several months following cessation of treatment. While this is truly a remarkable achievement many other similar cases have been treated unsuccessfully. The successful cases were discovered early and treated with large doses intramuscularly and intrathecally for many months.

Draining tuberculous sinuses treated with streptomycin usually respond favorably but there is a tendency to recurrence after treatment has been stopped.

Preliminary work with streptomycin in bone tuberculosis would suggest that it may prove to be a helpful adjunct to the orthopedic surgeons.

Tuberculosis of the skin treated with streptomycin has been helpful in some cases, and of dubious value in others. There is a tendency for the lesions to recur.

Very favorable results have been obtained in the treatment of tuberculous laryngitis and tracheo-broncheal tuberculosis. In these cases the drug has been given both intramuscularly and by inhalation (nebulization).

It must be borne in mind that streptomycin is *not* an over-night cure-all for the different types of tuberculosis in humans. Experience has already shown that. Many cases will receive no appreciable benefit from its use. It has limitations as do other valuable drugs such as penicillin and the sulfas. A vast amount of clinical research remains to be done before its assets and liabilities may be accurately determined. Many questions remain to be answered. For example: What is the optimum dose for a given case? How frequently should the drug be administered? What is the optimum duration of treatment? How will the development of drug fastness by the tubercle bacillus affect the dosage and duration of therapy in a given case? These are only a few of the problems requiring investigation.

The United States Public Health Service has taken a very keen interest in the therapeutic possibilities of streptomycin. A tuberculosis Study Section has been appointed recently by the Surgeon General to stimulate and correlate the clinical investigation of streptomycin and other drugs which may warrant study. It is hoped that federal funds will be appropriated to help finance this and other important research projects in the study of tuberculosis.

For the sake of the untold thousands suffering from tuberculosis, it is fervently hoped that its place in our armamentarium against this disease will prove to be a big one . . . and that the cost of streptomycin will be within the reach of all.

Committee on Chemotherapy and Allied Measures

Karl H. Pfuetze, M.D.
Cannon Falls, Minnesota
Chairman

Raphael A. Bendove, M. D.
New York, New York
Vice-Chairman

Reuben Hoffman, M.D.
Henrytown, Maryland

B. L. Freedlander, M.D.
San Francisco, Calif.

John V. Thompson, M.D.
Indianapolis, Indiana

Thirteenth Annual Meeting

AMERICAN COLLEGE OF CHEST PHYSICIANS

Ambassador Hotel, Atlantic City

June 5 - 8, 1947

P R O G R A M

THURSDAY, JUNE 5th

8:00 A. M. — Registration

9:00 A. M. — Administrative Sessions

Meeting, Executive Council

Meeting, Board of Governors

Meeting, Councils and Committees

9:00 A. M. — Examinations for Fellowship (Oral)

9:00 A. M. — Scientific Session

The Use and Abuse of Bed-rest in the Treatment of Tuberculosis
J. Winthrop Peabody, M.D., F.C.C.P., Professor, Diseases of the Respiratory System, Georgetown University School of Medicine, Washington, D. C.

Fifteen Years of Tuberculosis Control in a Rural Community
Lewis S. Jordan, M.D., Medical Director, Riverside Sanatorium, Granite Falls, Minnesota.

Progress in Tuberculosis Control in the United States
Francis J. Weber, M.D., Medical Director, Chief, Tuberculosis Control Division, U. S. Public Health Service, Washington, D. C.

Medical Aspects of Rehabilitation in Tuberculosis
Oscar Feinsilver, M.D., F.C.C.P., Director, Chest Clinic, Worcester City Hospital, Worcester, Massachusetts.

The Role of Bronchoscopy in Clinical Medicine and Surgery
Arthur Q. Penta, M.D., F.C.C.P., Visiting Lecturer, Temple University, Department of Medicine, Schenectady, New York.

12:00 Noon — Luncheon Meeting

Annual Conference of College Chapter Officials

Nelson W. Strohm, M.D., F.C.C.P., Buffalo, New York, Chairman.
Seymour M. Farber, M.D., F.C.C.P., San Francisco, Calif., Secretary.

2:00 P. M. — Meeting, Board of Regents

2:00 P. M. — Examinations for Fellowship (Written)

2:00 P. M. — Scientific Session

Extrapleural Pneumothorax

L. O. Davenport, M.D., F.C.C.P., Surgical Consultant, Tuberculosis Division, Alabama State Health Department, Louis L. Friedman, M.D., F.C.C.P., Assistant Professor of Medicine, University of Alabama, and Joseph C. Bohorfoush, M.D., F.C.C.P., Medical Superintendent, Jefferson County Tuberculosis Sanatorium, Birmingham, Alabama.

The Rationale of Therapeutic Pneumoperitoneum, Physiologic and Mechanical Considerations

Norman L. Anderson, M.D., F.C.C.P., Staff Member, The Norburn Clinic, Asheville, North Carolina.

Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis, A Clinical Study of 500 Cases

Benjamin L. Brock, M.D., F.C.C.P., Clinical Director, Veterans Hospital, and Torrence C. Moyer, M.D., F.C.C.P., Veterans Hospital, Oteen, North Carolina.

Combined Thoracoplasty

Gustave Maurer, M.D., F.C.C.P., Medical Superintendent, Schatzalp Sanatorium, Davos, Switzerland.

Surgical Treatment of Residual Cavities Following Thoracoplasties for Tuberculosis

J. D. Murphy, M.D., F.C.C.P., Chief, Surgical Service, Veterans Hospital, Oteen, North Carolina.

4:30 P. M. — Council and Committee Meetings

6:30 P. M. — Dinner, International Night

Harry C. Warren, M.D., F.C.C.P., San Francisco, California, Chairman, Council on Pan Pacific Affairs, presiding.

Andrew L. Banyai, M.D., F.C.C.P., Milwaukee, Wisconsin, Chairman, Council on European Affairs.

Chevalier L. Jackson, M.D., F.C.C.P., Philadelphia, Pennsylvania, Chairman, Council on Pan American Affairs.

A number of speakers from other countries are planning to come to Atlantic City to present interesting papers on the International Night program, including the following:

Extremely Conservative and Radical Treatment of Pulmonary Tuberculosis

Donato G. Alarcon, M.D., F.C.C.P., Director Medico, Sanatorio para Tuberculosis de la Asistencia Publica-Mexico, Director, Sanatorio San Angel, Professor de Clinica de Aparato Respiratosio de la Facultad de Medicina, Universidad Nacional, Mexico City, D. F.

Bronchiectasis in Children

Alberto Chattas, M.D., Jefe de Clinica, Instituto de Tisiologia, Jefe de Clinica, Maternidad Nacional, Cordoba, Argentina.

Bronchial Obstruction and Artificial Pneumothorax

Rene G. Mendoza, M.D., Hospital Sanatorio La Esperanza, Havana, Cuba.

Management of Pleural Effusions Secondary to Artificial Pneumothorax

David E. Garcia, M.D., F.C.C.P., Director, Clinica Fernandez Garcia, Hato Rey, Puerto Rico.

An Appraisal of the Results of a Six-Year Survey of Tuberculin Tests and Mass X-ray Examinations of the Population of Asuncion, Based on 130,000 Tuberculin Tests

Angel R. Gines, M.D., F.C.C.P., Eduardo Gould, M.D., and J. A. Coronel Vera, M.D., Asuncion, Paraguay.

Bronchography by Vaporization

Pedro L. Farinas, M.D., F.C.C.P., Director, Tecnico del Departamento al Rayos X, Hospital General Calixto Garcia, Havana, Cuba.

Tuberculosis Control in Australia; Present Conditions and Future Considerations

Sir Sidney Sewell, F.C.C.P., Honorary Physician, Royal Melbourne Hospital; Lecturer and Examiner in Medicine, Melbourne Medical School; Examiner for New Zealand University of Medicine and Dentistry, Melbourne, Australia.

FRIDAY, JUNE 6th

9:00 A. M. — Administrative Session

Report of the Secretary-Treasurer

Report of the Executive-Secretary

Reports of Councils and Committees

Election of Officers

New Business

12:00 Noon — Luncheon Meeting

Council on Public Health

Paul A. Turner, M.D., F.C.C.P., Louisville, Kentucky, Chairman.

Speakers representing the Army, Navy, Public Health Service and Veterans Administration will appear on this program.

2:00 P. M. — Scientific Session

Pulmonary Decortication

Thomas H. Burford, M.D., Associate Professor of Surgery, Washington University School of Medicine, Thoracic Surgeon, Barnes Hospital, Consultant in Thoracic Surgery, Robert Koch Hospital and U. S. Veterans Administration, St. Louis, Missouri.

Treatment of Patent Ductus Arteriosus

William S. Conklin, M.D., F.C.C.P., Assistant Professor of Surgery, and Assistant Professor of Medicine, University of Oregon Medical School, Portland, Oregon.

Present Status of the Surgical Treatment of Carcinoma of the Lung

William F. Rienhoff Jr., M.D., F.C.C.P., Associate Professor of Surgery, Johns Hopkins University, Baltimore, Maryland.

Multiple Segmental Resection in the Treatment of Bronchiectasis

Richard H. Overholt, M.D., F.C.C.P., Clinical Professor of Surgery, Tufts College Medical School, and Chief of Thoracic Clinic and Consultant in Thoracic Surgery, Tumor Department, Boston Dispensary, Reeve H. Betts, M.D., F.C.C.P., Assistant Professor of Surgery, Tufts College Medical School, and Francis M. Woods, M.D., Boston, Massachusetts.

Surgery of the Esophagus

John H. Garlock, M.D., Clinical Professor of Surgery, New York Postgraduate Medical School, Columbia University, New York, N. Y.

8:00 P. M. — "Information Please in Diseases of the Chest"

Harold G. Trimble, M.D., F.C.C.P., Oakland, California, Moderator.

Panel of Experts:

Medicine

Hobart A. Reimann, M.D., Professor of Medicine, Jefferson University, Philadelphia, Pennsylvania.

Surgery

Brian Blades, M.D., F.C.C.P., Professor of Surgery, George Washington University Medical School, Washington, D. C.

Pathology

S. A .Levinson, M.D., Professor of Pathology, University of Illinois College of Medicine, Chicago, Illinois.

Bronchoscopy

Henry B. Orton, M.D., Professor of Laryngeal Surgery, New York Polyclinic Medical School, Newark, New Jersey.

Roentgenology

Eugene P. Pendergrass, M.D., Professor of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania.

Note: If you would like to direct a question to any of the above experts please send your questions to the Executive Offices of the American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Both the questions and answers of those selected will be published in the College Journal, "Diseases of the Chest." The Program Committee reserves the right to select the questions which are to be presented at the meeting.

SATURDAY, JUNE 7th

9:00 A. M. — Scientific Session

Meig's Syndrome

Irving F. Stein, M.D., Associate Professor of Obstetrics and Gynecology, Northwestern University, Chicago, Illinois.

Pulmonary Embolism as a Cause of Acute Coronary Insufficiency

Arthur M. Master, M.D., Assistant Professor of Clinical Medicine, Columbia University College of Physicians and Surgeons, New York, New York.

The Bromptom Hospital: A Centenary Review

Clifford Hoyle, M.D., Editor, British Journal of Tuberculosis and Diseases of the Chest, London, England.

Role of the Gastrointestinal Tract in the Production of Thoracic Symptoms

A. H. Aaron, M.D., Professor of Clinical Medicine, and Leon J. Leahy, M.D., Assistant Professor of Surgery, University of Buffalo School of Medicine, Buffalo, New York.

Prospects and Methods for Effective Tuberculosis Control

Herman E. Hilleboe, M.D., F.C.C.P., Assistant Surgeon General, U. S. Public Health Service, Washington, D. C.

12:00 Noon — Luncheon Meeting

Conference of Medical Directors and Superintendents of Tuberculosis Hospitals and Sanatoria

Benjamin L. Brock, M.D., F.C.C.P., Oteen, North Carolina, Chairman.

Pitfalls in Dealing with Cancer Statistics, Especially as Related to Cancer of the Lung

Guest Speaker: Madge Thurlow Macklin, M.D., Ohio State University, Columbus, Ohio.

Report of the Subcommittee on Standards

I. D. Bobrowitz, M.D., F.C.C.P., Otisville, New York, Chairman.

Report of the Subcommittee on Rehabilitation

Allan Hurst, M.D., F.C.C.P., Denver, Colorado, Chairman.

2:00 P. M. — Scientific Session

Cystic Disease of the Lung

Otto C. Brantigan, M.D., F.C.C.P., Associate Professor of Surgery, University of Maryland Medical School, Baltimore, Maryland.

Ayerza's Disease

Alberto C. Taquini, M.D., Director, Cardiologic Research Center, University of Buenos Aires, Buenos Aires, Argentina.

The Surgical Lesions of Pulmonary Coccidioidomycosis

S. J. Greer, Capt., MC, AUS, and John B. Grow, Col., MC, AUS, Fitzsimons General Hospital, Denver, Colorado.

Indications for Pulmonary Resection for Tuberculosis both by Lobectomy and Pneumonectomy

Lyman A. Brewer, M.D., Assistant Clinical Professor of Surgery, College of Medical Evangelists, and Frank S. Dolley, M.D., F.C.C.P., Associate Professor of Surgery, College of Medical Evangelists, Los Angeles, California.

6:30 P. M. — Convocation (Formal)

Major General S. U. Marietta, President-Elect, Washington, D. C., presiding.

Guest speaker to be announced

Awarding of Life Membership Certificates

Awarding of Fellowship Certificates

7:30 P. M. — Cocktail Party

8:00 P. M. — Presidents' Banquet (Formal)

Toastmaster

J. Winthrop Peabody, M.D., F.C.C.P., Washington, D. C.

President's Address

Charles M. Hendricks, M.D., F.C.C.P., El Paso, Texas.

College Award

Major General S. U. Marietta, F.C.C.P., Washington, D. C.

SUNDAY, JUNE 8th

9:00 A. M. — Scientific Session

The Clinical Significance of Pulmonary Hemorrhage: A Study of 1316 Patients with Thoracic Disease

Osler A. Abbott, M.D., F.C.C.P., Chief, Division of Chest Surgery, and Assistant Professor of Surgery, Emory University Medical School, Emory, Georgia.

Asbestosis

Kenneth M. Lynch, M.D., Professor of Pathology and Dean, Medical College of the State of South Carolina, Charleston, South Carolina.

Bagassosis

W. A. Sodeman, M.D., Assistant Professor of Medicine, Tulane University, New Orleans, Louisiana.

The Importance and Value of Bacteriologic Studies of Surgical Pathologic Specimens

Herman J. Moersch, M.D., Associate Professor of Medicine, University of Minnesota Medical School, and L. A. Weed, M.D., Mayo Clinic, Rochester, Minnesota.

Streptomycin Therapy in Tuberculosis

Paul Bunn, M.D., Associate to the Chief, Tuberculosis Division, Member, Streptomycin Committee, United States Veterans Administration, Washington, D. C.

12:00 Noon — Luncheon Meeting

National Conference of Tuberculosis Committees

James H. Stygall, M.D., F.C.C.P., Indianapolis, Indiana, Chairman.

Recent Advances in Vitamin Research as Related to Clinical Medicine

Guest Speaker: Tom D. Spies, M.D., Associate Professor of Medicine, University of Cincinnati, Cincinnati, Ohio, and Hillman Hospital, Birmingham, Alabama.

Discussion of Texas Plan for the organization of Tuberculosis Committees in the state medical societies

2:00 P. M. — Scientific Session

Pancreatic Insufficiency in Early Life, with Special Reference to Its Pulmonary Manifestations

Sidney Farber, M.D., Pathologist and Chairman, Division of Laboratories, Children's Medical Center, and Assistant Professor of Pathology, Harvard Medical School, Boston, Massachusetts.

Parasitic Infestations of the Lung

Thomas T. Mackie, M.D., Professor of Preventive Medicine, Wake Forest College, The Bowman Gray School of Medicine, Winston-Salem, North Carolina.

Clinical Application of Angiocardiography

Henry K. Taylor, M.D., Director, Department of Radiology, Goldwater Memorial Hospital, Welfare Island, New York, New York.

Volume Changes in the Pulmonary Blood Vessels in Relation to Artificial Relaxation Therapy

Charles C. Macklin, M.D., Professor of Anatomy and Histology, University of Western Ontario, London, Ontario, Canada.

Significance of Solitary Mass in the Lung

Edgar W. Davis, M.D., F.C.C.P., Professor of Thoracic Surgery, Georgetown University School of Medicine, and Roy G. Klepser, M.D., Instructor in Clinical and Thoracic Surgery, Georgetown University School of Medicine, Washington, D. C.

5:00 P. M. — Meeting, Board of Regents

8:00 P. M. — X-Ray Conference

Louis Mark, M.D., F.C.C.P., Columbus, Ohio, Moderator.

Cases to be presented should be problem cases in which a definite diagnosis has been established by examination of a surgical pathological specimen, bronchoscopic examination, post-mortem examination, or other laboratory studies.

Physicians who wish to present x-ray films should send their films, plus a resume of the history, physical findings, significant laboratory work and necropsy or surgical findings, to Dr. Andrew L. Banyai, Chairman of the Scientific Program Committee, Muirdale Sanatorium, Milwaukee 13, Wisconsin.

The Committee will have slides made of those films which are selected for the Conference. The slides will be projected on a large screen so that they will be easily visible to the entire audience. Your kind cooperation in forwarding the films and other data to the Chairman of the Program Committee, will be very much appreciated.

Note: Unless otherwise indicated, all papers are to be limited to twenty minutes.

ANNOUNCEMENT

The Board of Examiners plans to conduct the next oral and written examinations for Fellowship in the American College of Chest Physicians at Atlantic City, New Jersey, June 5, 1947. The oral examinations will be conducted in the morning and the written examinations will be held in the afternoon. Only physicians whose applications for Fellowship have been approved by the Board of Regents, will be eligible for this examination. If you desire to take the examination, please write to the Executive Secretary, 500 North Dearborn St., Chicago 10, Illinois.

College Chapter News

ARIZONA CHAPTER

The annual meeting of the Arizona Chapter of the College will be held at the Pioneer Hotel, Tucson, on May 6, 1947, one day prior to the meeting of the Arizona Medical Association. The following scientific program will be presented:

W. Bernard Yegge, M.D., F.C.C.P., Denver, Colorado
(1) Silicosis.
(2) Virus Pneumonia.

Paul C. Samson, M.D., F.C.C.P., Oakland, California
(1) The Care of Thoracic Injuries.
(2) The Management of Chronic Broncho-Pulmonary Suppuration.

William S. Conklin, M.D., F.C.C.P., Portland, Oregon
(1) Pulmonary Resection for Pulmonary Tuberculosis.
(2) Surgery in Congenital Malformations of the Heart and Great Vessels.

Howell Randolph, M.D., F.C.C.P., Phoenix, Arizona
(1) Certain Aspects of Bronchography.

D. W. Melick, M.D., Phoenix, Arizona
(1) Tumors of the Lung.

Albert Eckstein, M.D., Phoenix, Arizona
(1) Medical Treatment of Broncho-Pleural Fistula.

There will be an "Information Please" program held after dinner.

Dr. Charles A. Thomas, Tucson, is President of the Arizona Chapter and Dr. Bertram L. Snyder, Phoenix, and Dr. Leslie B. Smith, Phoenix, are Vice-President and Secretary-Treasurer, respectively, of the Chapter.

CALIFORNIA CHAPTER

Dr. John C. Sharp, Salinas, President of the California Chapter of the College has made the following committee appointments:

Membership Committee:
Dr. Rudolph H. Sundberg, San Diego, *Chairman*
Dr. William A. Cassidy, Livermore
Dr. James T. Harkness, Berkeley
Dr. Cabot Brown, San Francisco
Dr. Carl B. Howson, Los Angeles

Program Committee:
Dr. Joseph L. Robinson, Los Angeles, *Chairman*
Dr. Forrest G. Bell, San Francisco
Dr. Gordon A. Diddy, Ahwahnee
Dr. Jane Skillen, Olive View
Dr. David T. Proctor, Pasadena

Nominating Committee:
Dr. Edward W. Hayes, Monrovia, *Chairman*
Dr. Jacob J. Singer, Beverly Hills
Dr. Robert M. Peers, Colfax

Postgraduate and Undergraduate Medical Education:
Dr. Seymour M. Farber, San Francisco, *Chairman*
Dr. Edward W. Hayes, Monrovia
Dr. William L. Rogers, San Francisco
Dr. Frank S. Dolley, Los Angeles

MISSOURI CHAPTER

The Missouri Chapter of the College will meet jointly with the Missouri Chapter of the American Trudeau Society on Sunday, March 30th, in Kansas City. The following program will be presented:

"Some Problems in Unsatisfactory Pneumothorax"
John T. Kalish, M.D., Koch, Missouri.

"Mistaken Diagnosis of Tuberculosis"
Rubin H. Kaplan, M.D., F.C.C.P., Excelsior Springs, Missouri

"Phrenic Crush — Twenty Consecutive Cases"
Edward W. Laboe, M.D., Howell, Michigan

"Pulmonary Calcifications and Histoplasmosis"
M. L. Furcolow, M.D., and
H. L. Mantz, M.D., F.C.C.P., Kansas City, Missouri.

"Streptomycin Therapy in Bronchial Tuberculosis"
Arthur M. Olsen, M.D., Rochester, Minnesota.

"Epidermal Anaesthesia in Chest Surgery"
Y. F. Fujikawa, M.D., F.C.C.P., and
Charles A. Brasher, M.D., F.C.C.P., Mount Vernon, Missouri.

There will be an X-ray Conference in the evening at which Dr. W. W. Buckingham, F.C.C.P., Kansas City, will preside.

NEW JERSEY CHAPTER

The New Jersey Chapter of the College will hold its annual meeting in Atlantic City, on April 23rd. There will be an X-ray Conference conducted by Dr. Martin H. Collier, F.C.C.P., Grenloch, New Jersey, which will be followed by cocktails and luncheon. Dr. George G. Ornstein, F.C.C.P., New York City, will be the guest speaker. The title of his paper is "Present Status of Chemotherapy in Tuberculosis." After an open discussion of Dr. Ornstein's paper there will be a business meeting and election of officers for the Chapter. A brief talk will be given by the incoming President of the New Jersey Chapter.

OHIO CHAPTER

The Ohio Chapter of the College will hold its annual meeting at the Cleveland Hotel, Cleveland, on Wednesday, May 7th, in conjunction with the annual meeting of the Ohio State Medical Society, May 6, 7 and 8, 1947. The following program will be presented:

"Silicosis"
William J. Habeeb, M.D., F.C.C.P., Springfield, Ohio.

"Bronchiectasis"
Raymond C. McKay, M.D., F.C.C.P., Cleveland, Ohio.

"Pneumoperitoneum"
Frank Lande, M.D., McConnelsville, Ohio.

"Thoracic Surgery in the Hospital During 1945"
W. L. Potts, M.D., F.C.C.P., Columbus, Ohio.

"Procedures for Demonstrating Tubercle Bacilli in the State of Ohio"
R. J. Ritterhoff, M.D., Cincinnati, Ohio.

NORTH MIDWEST CHAPTER

The annual meeting of the North Midwest Chapter will be held Tuesday afternoon, July 1st, in Duluth, Minnesota, in connection with the annual meeting of the Minnesota State Medical Association. A splendid program on chest diseases is being arranged, but the final details have not been completed. The complete program will appear in the next issue of "Diseases of the Chest."

TEXAS CHAPTER

The Texas Chapter of the College will hold its annual meeting at the Baker Hotel, Dallas, on May 5, 1947, in conjunction with the annual meeting of the Texas Medical Association. Dr. R. G. McCorkle, M.D., F.C.C.P., San Antonio, President of the Texas Chapter, will preside and the following program will be presented:

Morning Session (Medical):

"Pulmonary Cavitation — Some Diagnostic Problems"
Walter C. Brown, M.D., F.C.C.P., Corpus Christi, Texas.
Discussion opened by:
H. Frank Carman, M.D., F.C.C.P., Dallas, Texas.

"Pulmonary Infarction"
John Chapman, M.D., F.C.C.P., Dallas, Texas.
Discussion opened by:
H. Frank Carman, M.D., F.C.C.P., Dallas, Texas.

"Skin Sensitivities Correlated with Lung Radiopathies"
Ray Boster, M.D., Sanatorium, and
Victor E. Schulze, M.D., San Angelo, Texas.
Discussion opened by:
David McCullough, M.D., F.C.C.P., Kerrville, Texas.

"Tuberculosis Among Workers in Dusty Trades"
Carl A. Nau, M.D., Galveston, Texas.
Discussion opened by:
Henry R. Hoskins, M.D., F.C.C.P., San Antonio, Texas.

"Pulmonary Embolism"
John A. Wiggins Jr., M.D., F.C.C.P., Fort Worth, Texas.
Discussion opened by:
Elliott Mendenhall, M.D., F.C.C.P., Dallas, Texas.

The Nominating Committee and the Executive Committee will convene during recess. The Executive Committee will discuss policies and plans of the Texas Chapter and Dr. R. G. McCorkle, President of the Chapter, will present a Report of the State of the Tuberculosis Problem in the State of Texas, State Institutions, Board of Health, Death Rate, General, etc.

Afternoon Session (Surgical):

"A Stab Wound of the Heart — A Case Report"
Edward W. Coyle, M.D., F.C.C.P., San Antonio, Texas.
Discussion opened by:
Robert B. Homan Jr., M.D., F.C.C.P., El Paso, Texas.

"Thoracoplasty — Report of Results of a Consecutive Series"
Robert B. Homan Jr., M.D., F.C.C.P., El Paso, Texas.
Discussion opened by:
John Roberts Phillips, M.D., F.C.C.P., Houston, Texas.

"Mediastinal Tumors"
John Roberts Phillips, M.D., F.C.C.P., Houston, Texas.
Discussion opened by:
Thomas Jones, M.D., F.C.C.P., Houston, Texas.

"Transthoracic Gastric and Esophageal Resections with Anastomosis"
James E. Dailey, M.D., F.C.C.P., Houston, Texas, and
Howard T. Barkley, M.D., Houston, Texas.
Discussion opened by:
Robert Shaw, M.D., Dallas, Texas.

Banquet:
R. G. McCorkle, M.D., F.C.C.P., San Antonio, Texas, President Texas Chapter, American College of Chest Physicians, Presiding.

Report of President of Texas Chapter of its activities and accomplishments in 1946-1947 and the state of the Tuberculosis Problem in the State of Texas.

Introduction of Charles M. Hendricks, M.D., F.C.C.P., El Paso, Texas, President of the American College of Chest Physicians.

"Chest Surgery in the Tuberculous Negro"
Guest Speaker:
David Harvey Shipp, M.D., F.C.C.P., Little Rock, Arkansas.

Business session and Election of Officers.

MARYLAND - DISTRICT OF COLUMBIA CHAPTER

The Second Annual Meeting of the Maryland - District of Columbia Chapter will be held at the Belvedere Hotel, Baltimore, Maryland, on April 21st. The Annual Meeting of the Medical and Chirurgical Faculty of Maryland will be held in Baltimore, April 22nd and 23rd.

Registration for the chapter meeting opens at 1:00 p. m. and the scientific session will open at 2:00 p. m. The following program will be presented:

"Sarcoidosis"
Sol Katz, M.D., Washington, D. C.
Discussion:
E. Howard Tonolla, M.D., F.C.C.P., Baltimore, Maryland.

"Congenital Atresia of the Esophagus"
Cameron Haight, M.D., Ann Arbor, Michigan.
Discussion:
O. C. Brantigan, M.D., F.C.C.P., Baltimore, Maryland.

"The Surgical Treatment of Pulmonic Stenosis"
Alfred Blalock, M.D., Baltimore, Maryland.
Discussion:
Edgar W. Davis, M.D., F.C.C.P., Washington, D. C.

"Chemotherapy of Tuberculosis"
John A. Kolmer, M.D., Philadelphia, Pennsylvania.
Discussion:
John C. Krantz, Jr., Baltimore, Maryland.

"Correlation of Various Branches of Medicine and Surgery as Related to Diseases of the Chest"
J. Winthrop Peabody, M.D., F.C.C.P., Washington, D. C.
Discussion:
Maurice C. Pincoffs, M.D., Baltimore, Maryland.

Business Meeting
William F. Rienhoff Jr., M.D., F.C.C.P., President, Maryland-District of Columbia Chapter, presiding.

Cocktail Party.

Annual Banquet.

X-Ray Conference
Russell H. Morgan, M.D., Baltimore, Maryland, Moderator.

ILLINOIS CHAPTER

The Illinois Chapter of the College will hold its annual meeting in Chicago on May 11th, just prior to the meeting of the Illinois State Medical Society, May 12 - 14, 1947.

SPRING MEETING DATES

College Chapters	State Medical Societies
M A R C H :	
Missouri Chapter, March 30th, Kansas City, Missouri.	Missouri State Medical Association, March 30 - April 2.
A P R I L :	
New Jersey Chapter, April 23rd, Atlantic City.	Medical Society of New Jersey, April 22 - 23 - 24.
Maryland-District of Columbia Chapter April 21st, Baltimore, Maryland.	Medical and Chirurgical Faculty of Maryland, April 22 - 23.
M A Y :	
Texas Chapter, May 5th, Dallas.	State Medical Association of Texas, May 5 - 8.
Arizona Chapter, May 6th, Tucson.	Arizona State Medical Association, May 7 - 10.
Ohio Chapter, May 7th, Cleveland.	Ohio State Medical Society, May 6 - 8.
Illinois Chapter, May 11th, Chicago.	Illinois State Medical Society, May 12 - 14.
New York Chapter, Buffalo.	New York State Medical Society, May 5 - 9.
California Chapter, Los Angeles.	California Medical Association, April 30 - May 3.
J U L Y :	
North Midwest Chapter, July 1, Duluth, Minnesota.	Minnesota State Medical Association, June 30 - July 2.

COUNCILS AND COMMITTEES

A number of councils and committees of the College to which new members have been appointed were published in the January-February issue of the journal, and we are pleased to list below the present membership of some additional councils and committees:

Membership Committee:

Roy A. Wolford, M.D., Washington, D. C., *Chairman*
Capt. Robert E. Duncan, USN, Washington, D. C., *Vice-Chairman*
Maj. Gen. S. U. Marietta, Washington, D. C.
Leo Eloesser, M.D., San Francisco, California
Herman E. Hilleboe, M.D., Washington, D. C.
R. B. Homan, M.D., El Paso, Texas
Chevalier L. Jackson, M.D., Philadelphia, Pennsylvania
William E. Ogden, M.D., Toronto, Canada
Richard H. Overholt, M.D., Brookline, Massachusetts
Joseph C. Placak, M.D., Cleveland, Ohio

Committee on Chest Diseases in Institutions:

Otto L. Bettag, M.D., Pontica, Illinois, *Chairman*
B. B. Bagby Jr., M.D., Martinsburg, West Virginia
G. C. Bellinger, M.D., Salem, Oregon
H. A. Burns, M.D., St. Paul, Minnesota
George Curtis, M.D., Columbus, Ohio
A. A. Leonidoff, M.D., Poughkeepsie, New York

Committee on State Laws for Tuberculosis:
(Subcommittee of Council on Public Health)

Andrew L. Banyai, M.D., Milwaukee, Wisconsin, *Chairman*
Joseph E. Blum Jr., M.D., Greenwell Springs, Louisiana
Willard B. Howes, M.D., Detroit, Michigan
J. George Lang, M.D., New York, New York
William F. Wagner, M.D., San Francisco, California

Veteran Medical Officers Committee:

Philip H. Narodick, M.D., Seattle, Washington, *Chairman*
Osler A. Abbott, M.D., Emery, Georgia
Hyman E. Bass, M.D., New York, New York
Abel Froman, M.D., Chicago, Illinois
David H. Shipp, M.D., Little Rock, Arkansas
John W. Stacey, M.D., Los Angeles, California

COUNCIL ON PUBLIC HEALTH

Dr. Paul A. Turner, Louisville, Kentucky, Chairman of the Council on Public Health of the College, announces that a meeting of the Council took place in Washington, D. C. on February 27th. The program to be undertaken by the Council was fully discussed and plans were made for the luncheon meeting sponsored by the Council which is scheduled to be held on Friday, June 6th, in Atlantic City, in connection with the 13th Annual Meeting of the College. A full report will be given at that time.

Those attending the meeting in Washington were Comdr. Sidney A. Britten, Washington, D. C., Dr. Herman E. Hilleboe, Washington, D. C., Dr. Walter E. Vest, Huntington, West Virginia, who are members of the Council, Dr. Paul A. Turner, Louisville, Kentucky, Chairman, and, by invitation, Dr. John Barnwell, Major General S. U. Marietta, and Dr. J. Winthrop Peabody, Washington, D. C.

College News Notes

DELEGATES TO LIMA CONGRESS

The following Fellows of the American College of Chest Physicians will be delegates to the 7th Pan American Congress on Tuberculosis being held at Lima, Peru, March 17-22, 1947: Dr. Chevalier L. Jackson, Philadelphia, Pennsylvania, Chairman, Council on Pan American Affairs; Dr. Jay Arthur Myers, Minneapolis, Minnesota, Editor, "Diseases of the Chest"; Dr. Richard H. Overholt, Brookline, Massachusetts, First Vice-President; Dr. J. Winthrop Peabody, Washington, D. C., Chairman, Council on Postgraduate Medical Education; and Mr. Murray Kornfeld, Chicago, Illinois, Executive Secretary.

Mr. Kornfeld will visit a number of the Central American countries enroute to Lima and following the Congress he will visit Chile, Argentina, Uruguay and Brazil in the interests of the American College of Chest Physicians.

The Milwaukee Metropolitan Section of the American College of Chest Physicians met at the Medford Hotel on Friday, December 27, 1946. Dr. Valentine O'Malley spoke on Tracheobronchial Tuberculosis.

The Rocky Mountain Chapter had a special meeting and luncheon for Dr. Paul H. Holinger, F.C.C.P., of Chicago at the Cosmopolitan Hotel, Friday, February 21st, during the mid-winter clinics.

Dr. Alton Ochsner, F.C.C.P., New Orleans, Louisiana, received the Times Picayune Loving Cup for cumulative community service. The award of this loving cup to Dr. Ochsner is well merited. He has been an outstanding man in his community since he became Professor of Surgery at Tulane in 1926.

Dr. W. Bernard Yegge, F.C.C.P., Denver, Colorado, Secretary-Treasurer of the Rocky Mountain Chapter of the College, was elected to the office of President-Elect for the Medical Society of the City and County of Denver, to serve during 1948.

The Board of Regents of the University of California has notified the Dean of the Medical School that an anonymous donor has given $5,000 for a study on Primary Cancer of the Lung. The study will be directed by Dr. Seymour M. Farber, F.C.C.P., San Francisco, Secretary-Treasurer of the California Chapter of the College.

Dr. Fred Meixner, F.C.C.P., Peoria, Illinois, Ex-Regent of the College, and Past-President of the Illinois Chapter, has been appointed to the Medical Staff of the Peoria Municipal Tuberculosis Sanitarium. Dr. Dan Morse, F.C.C.P., Peoria, Illinois, is Medical Director.

The Mexican Chapter of the College has notified the Executive Secretary that a large delegation of members from Mexico will attend the 13th Annual Meeting of the College in Atlantic City, June 5 - 8, 1947. The delegation will be headed by Dr. Donato G. Alarcon, Mexico City, Regent of the College for Mexico, and will include, among others, Drs. I. Cosio Villegas, Vice-President of the Mexican Chapter, Manuel Alonso, Secretary of the Chapter, Miguel Jimenez, Fernando Rebora, and Jesus Benitez.

ANNOUNCEMENT

The Annual Meeting of the American Broncho-Esophagological Association will be held at the Hotel Brighton, Atlantic City, New Jersey, June 9, 1947.

Obituary

WILLIAM S. CLARK, III

1909 - 1946

Dr. William A. Clark, III, was born on April 7, 1909 in New Wilmington, Pennsylvania. After completing his high school education in that city he attended Westminster College for part of his pre-medical education. He entered Ohio State University in 1930 and completed his pre-medical education at that school. In 1932 he entered the Medical College of Ohio State University and graduated in 1936 with honors.

He served his internship at Ohio State Sanatorium in Mt. Vernon, Ohio, and at Saginaw County Hospital in Saginow, Michigan. He came to the Clark County Tuberculosis Sanatorium in Springfield, Ohio, as a resident in 1938 and spent four years in this institution as a resident. He was then promoted to superintendent of the sanatorium and served in this capacity for the next three years. In 1944 he entered private practice in Springfield, Ohio, and limited his practice to diseases of the chest.

Dr. Clark was on the active medical staff in the Springfield City Hospital at the time of his death. He was a member of the American Medical Association, Clark County Medical Society, and the American College of Chest Physicians.

He was married in 1932 and had two children, William A. Clark, IV, and Vicki, ages 12 and 8, respectively.

He was loved and respected by all of his patients and was held in high esteem by his fellow physicians. His early death at the age of 37 was a distinct loss to the entire community.

Lynne E. Baker, M.D., F.C.C.P., Dayton, Ohio.

MEMBERS OF THE COLLEGE RELEASED FROM THE ARMED SERVICES

Adelman, Louis, Major, MC, 175 Wilson Ave., Columbus, Ohio.

Batory, Roman J., Captain, MC, Route 3, Stroudsburg, Pa.

Bell, Forrest Gunn, Lt. Col., MC, Veterans Administration Branch Office No. 12, San Francisco 5, California.

Beverly, Squire, Lt. Col., MC, Veterans Administration Facility, Rutland Heights, Massachusetts.

Blitz, Oscar, Colonel, MC, 1504 Foucher Street, New Orleans 15, Louisiana.

Bruce, Paul C., Colonel, MC, Veterans Administration, Legion, Texas.

Callander, Russell John, Comdr., MC, Mountain Home, Tennessee.

Camp, Milton N., Major, MC, 311 S. Andrews Ave., Ft. Lauderdale, Florida.

Chodoff, Richard J., Major, MC, 1726 Pine Street, Philadelphia, Pa.

Craft, Charles B., Captain, MC, 19 W. Babcock St., Bozeman, Montana.

Daniels, Samuel D., Major, MC, 1260 S. Hayworth Ave., Los Angeles, Calif.

Diamond, Sidney, Captain, MC, 89 Hollis Court Boulevard, Queens Village New York.

Dierolf, Edward J., Captain, MC, 504 Broadway, Gary, Indiana.

Dorfmann, Henry L., Lt., MC, 50 Park Avenue, New York 16, N. Y.

Farness, O. J., Lt. Col., MC, 721 N. Fourth Ave., Tucson, Arizona.

Fishbein, Elliott, Captain, MC, Valley View Sanatorium, Paterson, N. J.

Fitzgerald, Joseph M., Lt., MC, USNR, 206 10th St., Hoboken, N. J.

Garment, Edward M., Major, MC, U. S. Veterans Hospital, Castle Point, New York.

Gaynor, John S., Captain, MC, 75 Twelfth Street, Wheeling, W. Virginia.

Gellman, Isaac Irving, Major, MC, Veterans Administration Facility, Huntington, West Virginia.

Goen, Rayburne W., Major, MC, 7 West Sixth Street, Tulsa 3, Oklahoma.

Heaton, Thomas Gilbert, Lt. Col., MC, 210 St. Clair Avenue, West, Toronto, Ontario, Canada.

Hughes, Felix A. Jr., Lt. Col., MC, 636 Patterson St., Memphis 15, Tenn.

Ivey, Hubert T., Lt. Col., MC, Veterans Administration, Legion, Texas.

Keiserman, Joseph, Captain, MC, 5460 Baltimore Ave., Philadelphia, Pa.

Kennedy, William M., Major, MC, Essex County Sanatorium, Verona, N. J.

Kilpatrick, Elmer Martin, Major, MC, 141 East Second South, Salt Lake City, Utah.

Knight, John S., Commander, MC, 5800 High Drive, Kansas City, Mo.

Kough, Othello S., Captain, MC, 36 W. Church St., Uniontown, Pa.

Leslie, George Lawrence, Lt. Col., MC, 440 W. Huron St., Pontiac, Mich.

Levin, Louis, Major, MC, Veterans Administration Facility, Excelsior Springs, Missouri.

Libien, Benjamin H., Captain, MC, 5302 Fifteenth Ave., Brooklyn, N. Y.

Lufkin, C. Dexter, Lt. Comdr., MC, Tuberculosis Division, Veterans Administration, Minneapolis, Minnesota.

Mathis, J. A., Lt. Comdr., MC, 240 S. Walnut St., Pinckneyville, Illinois.

Mayne, Roy Malone, Comdr., MC, 3028 E. Superior St., Duluth 5, Minn.

McHugh, John B., Major, MC, Finney Veterans Hospital, Thomasville, Ga.

Miller, Benjamin, Captain, MC, 810 Shasta St., Yuba City, California.

Newcomer, William, Lt. Comdr., MC, Veterans Administration Hospital, Oteen North Carolina.

Oppenheim, Heinz, Lt., MC, EENT Clinic, Crile V. A. Hospital, Cleveland 9, Ohio.

Pesquera, Gilberto S., Major, MC, Metropolitan Life Insurance Co., 1 Madison Avenue, New York, New York.
Schneider, Leo V., Lt. Col., MC, Glenn Dale Sanatorium, Glenn Dale, Md.
Schwartz, Raymond L., Major, MC, 1015 N. Highland St., Arlington, Va.
Smith, Jacob, Lt., MC, 12 East 76th St., New York 21, New York.
Smith, Leslie Benjamin, Lt. Col., MC, 505 W. Cypress, Phoenix, Arizona.
Solomon, Saul, Major, MC, 162 W. 54th St., New York, New York.
Sparer, Phoneas J., Captain, MC, 4000 E. 6th Ave., Denver, Colorado.
Steer, Albert Elon, Major, MC, 405½ S. 6th St., Springfield, Illinois.
Stephenson, Martin Lee Jr., Captain, MC, Apt. 65-C, Waterman Park, Fairfield, California.
Thomas, David R. Jr., Captain, MC, 924 Hickman Rd., Augusta, Georgia.
Titche, Leon A., Major, MC, 1701 E. Linden Ave., Tucson, Arizona.
Tyson, M. Dawson, Lt. Col., MC, 8 Occum Ridge, Hanover, N. H.
Watson, William L., Lt. Comdr., MC, 1088 Park Avenue, New York 28, N. Y.
Weiss, Alter, Captain, MC, 425 West End Ave., New York, New York.
Wheir, William Hugh, Lt. Comdr., MC, Box 430, Amarillo, Texas.
Wickler, Gerhard S., Lt., MC, Fort Bayard, New Mexico.
Williams, M. M., Captain, MC, Minnesota State Sanatorium, Ah-Gwah-Ching, Minnesota.
Wood, Kenneth A., Lt., MC, 2206 David Broderick Tower, Detroit, Mich.

NOTE: For additional list of members released from services see the November-December 1945, March-April 1946, and January-February 1947 issues of the journal.

READER NOTICE

New Penicillin Salt Broadens Therapeutic Applicability

Announcement was recently made by Commercial Solvents Corporation of a new penicillin salt which greatly broadens the applicability of antibiotic therapy. This new preparation is the crystalline potassium salt of penicillin G, the most effective penicillin species available.

Crystalline potassium penicillin is especially useful when the administration of sodium salts is contraindicated, as for example in edematous states and in certain forms of chronic nephritis.

This new penicillin salt is high in purity, providing not less than 1435 units per milligram. It is heat-stable, hence does not require refrigeration.

This new penicillin salt is available in crystalline form for parenteral use, in tablets buffered for oral use, and in a Romansky Type Formula.

Because it is odorless and practically tasteless, it is preferred by the patient when used in aerosol therapy. In addition, because of its high degree of purity, it can be employed in high concentration without producing signs of upper respiratory tract irritation.

In the Romansky type formula, the use of the potassium salt presents several advantages over the usual amorphous calcium salt. This preparation, white in color, is nonirritating on injection; according to all available reports, it produces no local reaction or persistent nodulation and gives assayable blood levels in most cases for 24 hours from a single 1 cc. dose containing 300,000 units.

Commercial Solvents Corporation was the first to produce crystalline penicillin commercially, and again first in producing this new potassium salt.

Positions Wanted and Available

MEDICAL SERVICE BUREAU

In accordance with a resolution adopted by the Board of Regents of the College at their annual meeting held in Chicago on June 17, 1945, a Medical Service Bureau has been established at the Executive Offices of the College for the purpose of serving the members of the College being released from the armed forces.

The Bureau would appreciate receiving information from the medical superintendents of sanatoria regarding positions available at their institutions, together with full particulars as to the type of position and salary offered. Fellows of the College who are looking for assistants should send complete information to the Bureau.

Physicians being released from the armed forces who are seeking appointments and positions should send complete information to the Bureau regarding their training and the type of position desired.

Please direct all correspondence to the Medical Service Bureau, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

POSITIONS AVAILABLE

Assistant physician wanted in tuberculosis hospital in southeast Texas. Three thousand a year with complete maintenance to single man. Excellent opportunity to learn chest surgery and other collapse therapy. Good climate the year around. For further information please write Box 148A, American College of Chest Physicians, 500 N. Dearborn Street, Chicago 10, Illinois.

Staff physician wanted at approved hospital, complete medical and surgical service, out-patient facilities, tuberculosis and other chest diseases. For further information please write Box 149A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Single doctor wanted as resident physician in tuberculosis sanatorium. For further information please write Box 151A, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Illinois.

Resident physician wanted at approved tuberculosis sanatorium for the training of residents in tuberculosis. Salary offered will depend on the experience and qualifications of applicant. Applicants should give their training, experience, age, etc. in their first letter and, if possible, enclose a photograph. Please address Box 152A, American College of Chest Physicians, 500 North Dearborn St., Chicago 10, Illinois.

Physician wanted for medical staff of the State Tuberculosis Sanatorium, Marianna, Florida, opened September 2, 1946, with 200 bed capacity. The salary is $300.00 or more depending upon experience, with full maintenance for self and family. Please address Dr. W. D. Rosborough, Superintendent and Medical Director of the sanatorium.

POSITIONS WANTED

Fellow of the College, with 15 years full time hospital and sanatorium experience in tuberculosis and chest diseases, desires Medical Directorship in progressive institution with good clinical opportunities. For additional information please address Box 232A, American College of Chest Physicians, 500 North Dearborn St., Chicago 10, Illinois.